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TREATMENT OF CANCER OF CERVIX BY RADIUM AND DEEP X-RAYS*

Experience at the Rhode Island Hospital, 1933-1943

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THE purpose of this paper is to report to the New England Surgical Society the results of treatment of cancer of the cervix at the Rhode Island Hospital from 1933 through 1943. The year 1933 is chosen as a starting point because in that year the use of deep x-ray therapy as a part of the "planned treatment" was inaugurated.

Four hundred and thirty-two consecutive cases of cancer of the cervix are found in the clinical files for this period. From a study of five-year and ten-year survival rates in this series of cases, we hope to draw conclusions regarding the effectiveness of methods of radiologic treatment on the group as a whole, with particular emphasis on its value in the treatment of early cases.

In this report the rules, definitions and classification by stages adopted in 1937 by the Committee on Cancer of the Cervix of the League of Nations Health Organization have been followed.[1] The tables presented are simplified and condensed from those prescribed for use in reporting series by collaborating clinics. On invitation the Rhode Island Hospital Clinic has become a collaborator this year. The complete tables have been forwarded to the League of Nations Committee for inclusion in their next annual report.

TABLE 1. *Patients Examined at the Clinic.*

YEAR	TOTAL NO. OF PATIENTS EXAMINED WITH A VIEW TO TREATMENT	PATIENTS EXAMINED AT THE CLINIC BUT NOT TREATED	
		NO.	PERCENTAGE
1933	54	2	3.6
1934	38	0	0
1935	31	0	0
1936	35	3	8.6
1937	38	1	2.6
1938	40	1	2.5
1939	35	2	5.7
1940	34	2	5.8
1941	33	2	6.1
1942	52	1	1.9
1943	42	1	2.4
Totals	432	15	
Average			3.7

METHOD

The method of treatment of cancer of the cervix with long element needles and deep x-ray therapy in use by this clinic has been adequately described in previous publications.[2-7] Complications have been reviewed,[8] and the place of deep x-ray therapy discussed in separate papers.[9] These subjects are not repeated in this paper.

Briefly, the method is based on the sound radiologic principles of multiple low-intensity foci, platinum filtration (gamma radiation) and long (168-hour) interval. The needles are element needles guided into place about the cervix, as nearly as can be determined by palpation to the advancing zone of the growth. A 20-mg. platinum-filtered capsule is also placed in the cervical canal. Deep x-ray treatment is given to the patients in Stage I approximately eight weeks after needle treatment. Most patients in Stage II are similarly treated. In this way the maximum effect on the primary tumor can be obtained with radium, and the x-ray treatment is then given eight weeks later. The principal objective of the x-ray therapy is to cover the lateral parametria and pelvic side walls. Two large fields, 15 cm. square, anterior and posterior, are used with a lead strip to protect as much as possible the previously irradiated cervix and paracervical area. In the badly infected and advanced cases (Stage III and IV) x-ray therapy comes first without lead strip and is effective in clearing up infection and preparing for radium later. In these cases with the cervix and paracervical tissues already heavily irradiated, further treatment with radium should be modified by the use of fewer needles and possibly by reduction in the time of treatment lest overdosage, with serious complications, result.

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Results

Table 1 demonstrates that there were 432 patients examined with a view to treatment. Of these, 15 were not treated. These included 12 patients in whom extensive disease made treatment unsuitable even for palliation, 2 who refused treatment and 1 who sought treatment elsewhere. In other words, 3.7 per cent were not treated, or 96.3 per cent of the patients applying, were accepted for treatment.

Table 2 gives the division of cases according to the extent of disease as defined in the 1937 classification of the Committee. Briefly, the classification is as follows:

Table 2. *Classification of Cases According to Stage of Disease.*

Year	Total No. of Patients	Patients in Stage I	Patients in Stage II	Patients in Stage III	Patients in Stage IV
1933	52	18	27	4	3
1934	38	10	18	6	4
1935	31	10	18	3	0
1936	32	10	12	4	6
1937	37	11	23	3	0
1938	39	15	21	1	2
1939	33	8	19	6	0
1940	32	14	13	5	0
1941	31	12	13	3	3
1942	51	16	31	3	1
1943	41	16	13	3	9
Totals	417	140	208	41	28
Percentages	100	34	50	10	6

Stage I. Disease confined to the cervix.

Stage II. Disease extends to the fundus, to the parametria short of fixation of the pelvic wall or to the upper two thirds of the vagina.

Stage III. Disease extends to the lateral pelvic wall, with fixation on one or both sides, to the lower third of the vagina. Palpable metastatic nodes are present on the pelvic walls.

Stage IV. Disease invades the bladder or rectum, vesicovaginal fistula is present, or disease extends beyond the true pelvis, with distant metastases.

There were 417 cases treated; 140, or 34 per cent of the total, were classified as Stage I, 208, or 50 per cent of the total, were Stage II, 41, or 10 per cent, were Stage III, and 28, or 6 per cent, were Stage IV.

In Figure 1 the survival-rate curves for each stage and the group as a whole are presented for periods of one to five years. The composite curve for the whole group shows that of the total of 417 patients seen and treated between 1933 and 1943, 170, or 40.7 per cent, were alive without evidence of disease at the end of the five years of observation. This represents the relative cure rate. The "absolute cure rate" for this group is 39.4 per cent and is based on the total of 432 patients examined with a view to treatment. In Stage I, representing 34 per cent of the total, 70 per cent survived for five years. In Stage II, which represented 50 per cent of the total, 33.2 per cent survived for five years. In Stage III, representing 10 per cent of the total, only 7.3 per cent of the patients survived for five years or more, and in Stage IV, representing 6 per cent of the total, there were no five-year survivors. The outcome in the 247 cases not included as five-year cures was as follows: death from cancer, 221, death from intercurrent disease, 22, survival with demonstrable cancer, 3, and lost sight of, 1.

Evaluation of results in the treatment of cancer necessarily depends on the adequate follow-up study of a high percentage of cases. In this series, 162 of 170 patients reported as five-year survivals were seen and examined regularly by members of the Tumor Clinic staff. Eight of the 170 patients, although not seen for one to four years, have been communicated with and are known to be alive and free of symptoms.

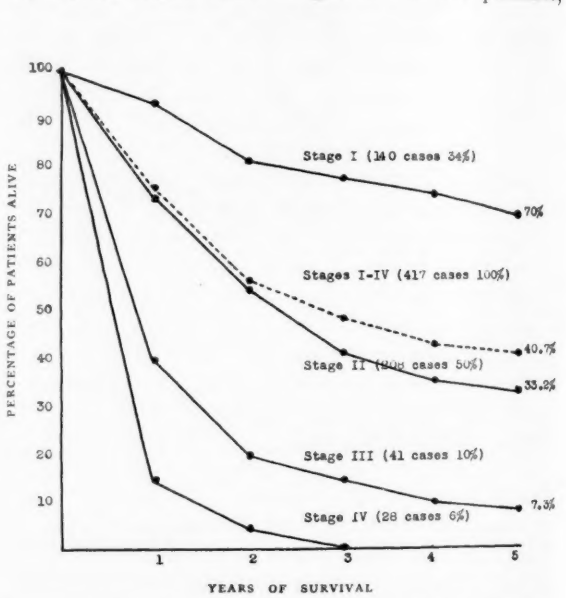

Figure 1. *Five-Year Follow-up Study (Relative) of 417 Cases of Carcinoma of the Cervix at Rhode Island Hospital, 1933-1943.*

In Figure 2 survival rates are presented for one to ten years in 229 cases seen and treated during the years from 1933 to 1938. Of the 229 cases treated, 74, or 32 per cent, were in Stage I, and these had a five-year survival rate of 66 per cent and a ten-year survival rate of 56 per cent. There were 119 cases, or 52 per cent of the total, in Stage II. These had a 30 per cent five-year and a 25 per cent ten-year survival rate. Twenty-one, or 9 per cent of the total, were in Stage III. Five per cent of these lived for five years; there were no ten-year survivals in this group. Cases in Stage IV comprised 7 per cent of the total, and there were no five-year survivals. The composite curve for the whole group shows that the five-year relative cure rate for the 229 cases was 36.6 per cent and the ten-year relative cure rate 31 per cent.

Comparison of rates for each stage between Figures 1 and 2 shows improvement in five-year cures for the whole group, 1933-1943, over the 1933-1938 group. It should be noted that there was a fall of 5.6 per cent in relative cure rate between the fifth and tenth years. Of 84 patients alive at the five-year interval, 70, or 84 per cent, survived for ten years.

Discussion

The Rhode Island Hospital Clinic seems fortunate in having a relatively high percentage of patients with favorable cases presenting themselves for treatment. Thirty-four per cent of the treated cases were placed in Stage I, and 50 per cent in Stage II. These percentages for Stage I and II are the highest presented by any of the collaborating clinics in the 1948 *Bulletin of the League of Nations Health Organization*. Conversely, the percentages of Stage III and IV were among the lowest presented.

Comparison of material between different clinics is obviously difficult. Undoubtedly, some clinics are dumping grounds for advanced or even terminal cases, and others in more favorable locations receive a higher percentage of earlier cases. Differences in interpretation of definitions can also affect the classification. Some operators who have a more pessimistic outlook tend to be influenced toward the darker side, whereas others who are more optimistic tend toward the lighter side.

The Rhode Island Hospital is situated in a thickly populated, industrial area. The hospital is the largest in Rhode Island, with a bed capacity of over 500, and, although privately owned and operated by a corporation, has served for many years as the large charity hospital for the city of Providence and the state of Rhode Island and is obligated to receive for treatment all patients applying. Therefore, the hospital receives the largest proportion of patients with cancer in the community. The relatively high proportion of early cases is apparently not due to selection.

Within a few years of the inception of the Gynecological Tumor Clinic at the Rhode Island Hospital in 1922, quite a distinct improvement in the type of cases at the time the patient sought treatment took place. The incidence of early cases rose from 19 per cent in 1921-1925 to 34 per cent in 1926-1928. The cases in Stage IV dropped from 45 per cent in 1921-1925 to 28 per cent in 1926-1928, and the 1934-1938 series shows only 18.8 per cent of cases in Stage IV. This improvement, we believe, is due to the influence that the Clinic has exerted on lay and professional education in the early diagnosis of cancer of the female reproductive organs in Rhode Island during those years.

The relative survival rate of 70 per cent in the cases in Stage I, comprising 34 per cent of the total group, seems very encouraging for this method of radiologic treatment. Regardless of the fact that the Clinic has favorable material, the results are comparatively high in this group. The low fall in survival rates between the fifth and the tenth year (5.6 per cent), uncorrected for patients dying of intercurrent disease (7 of 14), speaks well for the permanency of cure by this method.

Summary and Conclusions

A group of 432 consecutive cases of cancer of the cervix is presented.

Five-year and ten-year absolute and relative salvage rates are given.

The comparatively high incidence of early cases in this clinic is commented upon, and the comparatively high salvage rate in this early group noted.

Because of the favorable results, it is concluded that early cancer of the cervix is best treated by radium and x-ray therapy in this clinic.

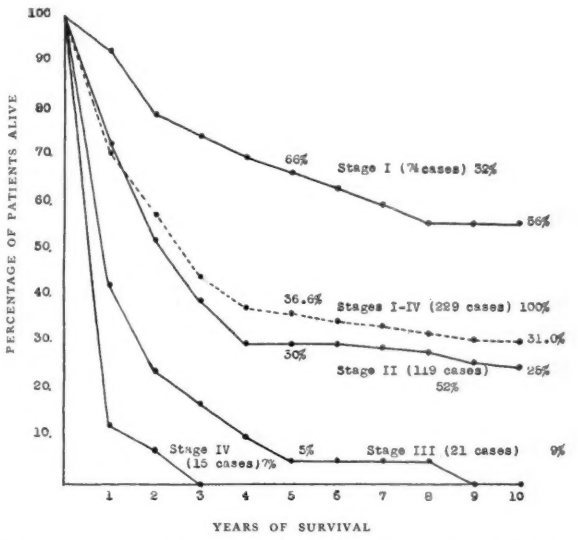

Figure 2. *Ten-Year Follow-up Study of 229 Cases of Carcinoma of the Cervix at Rhode Island Hospital, 1933-1938.*

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THE CLINICAL RESPONSE OF RHEUMATIC FEVER AND ACUTE CARDITIS TO ACTH (Concluded)*

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RESULTS

Nearly all the important manifestations of rheumatic fever are represented by the 10 patients whose case reports have been summarized. At the time ACTH therapy was begun, 8 patients had definite fever, 7 had symptoms or signs — or both — referable to the joints, 3 had subcutaneous nodules, 1 had chorea, 9 had signs of valvular involvement, 4 had pericarditis, 4 were in congestive failure, and all but 1 had elevation of the erythrocyte sedimentation rate. It is also evident from the case reports that the majority of the patients suffered from severe rheumatic fever and a high degree of active rheumatic carditis.

The marked variability in the course of rheumatic fever, when it is uninfluenced by therapy, necessitates the postponement of final judgment regarding the value of any form of treatment until there has been an opportunity to study a large series of patients over a long period. Nevertheless, the response of most of the patients treated with ACTH has been such that it is difficult to disregard the indications that this hormone often causes a marked suppression of rheumatic activity and even shortens the course of the disease in some cases.

Of the 8 patients in whom therapy has been completed, 5 (Cases 1, 4, 6, 7, and 8) showed striking improvement. All these patients became markedly better within the first few days or the first week of treatment, and the rheumatic process, as far as could be determined, became completely quiescent by the end of four weeks in Cases 6 and 8, six weeks in Case 7, eight weeks in Case 4 and ten weeks in Case 1. The 2 patients who are still under treatment (Cases 9 and 10) also showed a remarkably good immediate response to ACTH, but it is too soon to assess the final results of therapy in these cases.

The patient in Case 2 was extremely ill prior to ACTH therapy, with very active rheumatic fever, severe long-standing rheumatic heart disease and congestive failure. Her immediate response was impressive, and now, although low-grade rheumatic fever is probably still present and although her previously existing severe heart damage remains unchanged, her general condition contrasts quite strikingly with that before hormone treatment.

A patient with severe pancarditis (Case 5) responded more slowly to ACTH. Nevertheless, at the present time all evidence of active rheumatic fever has finally disappeared, and, considering the severity of her original illness, her course has not been unsatisfactory.

The only patient in whom a favorable response to ACTH could not be demonstrated is Case 3, a seven-year-old girl who was extremely ill with subcutaneous nodules, severe valvular damage and cardiac enlargement, and congestive failure. Because of the rapid fluctuation of this patient's clinical status prior to ACTH therapy her subsequent course has been somewhat difficult to evaluate. Nevertheless, the slowness and questionable completeness with which hepatic enlargement disappeared, the persistence of pallor and appearance of poor health, and the recent development of new subcutaneous nodules strongly suggest that active rheumatic fever has not yet subsided.

In general, then, it is evident that the clinical response was quite satisfactory in all but 1 of the 10 patients, but that the speed and extent of their response varied considerably. It is not possible from the available data to determine whether the clinical variations observed were due to differences in ability of the various patients to respond to the hormone or to differences in duration of treatment and variations in individual and total daily dosage of ACTH.

There is, of course, as yet no reason to believe that patients who have recovered from an attack of rheumatic fever after ACTH therapy are any less likely to have rheumatic recurrences when exposed to the hemolytic streptococcus than patients who have spontaneously recovered from this disease.

Response of Various Rheumatic Manifestations to ACTH

The effect of ACTH on each of the various rheumatic manifestations is of interest and requires separate consideration. Table 1 indicates the number of days required for the more important symptoms and signs to disappear in each of the 10 patients. Attention should be called to the fact that

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the figures in the table refer to the time for initial disappearance. Certain rheumatic manifestations reappeared in some cases, usually when ACTH was reduced in dosage or entirely omitted. These cases are discussed below more fully.

Of all manifestations fever and polyarthritis came under control the most rapidly. Definite elevation of the temperature, which was present in 8 cases, subsided within twenty-four hours in all but 1, in which it disappeared after forty-eight hours of treatment. An interesting phenomenon observed in several cases during the first one to three weeks of therapy was the maintenance of the temperature between 98 and 99°F. by rectum — a level that is somewhat lower than that usually encountered in normal children and in patients with rheumatic fever who have reached the quiescent stage of their disease.

Objective or subjective arthritis (or both) was present at the time ACTH treatment was begun in 7 cases and completely cleared up within one or two days in 5 of these. The remaining 2 patients, although much improved within twenty-four hours, required three or four days before their joints became entirely normal.

Pericarditis was present in 4 patients (Cases 1, 5, 7 and 9). In 3 of these (Cases 1, 5 and 9) there was also sufficient pericardial effusion to cause pulmonary compression signs over the left side of the chest posteriorly. All physical signs of pericardial involvement disappeared within twenty-four hours of therapy in Case 7 and within three days in Case 1. The patient in Case 5, whose slow general response to ACTH therapy has already been mentioned, required eleven days before the pericardial friction rub and pulmonary compression signs completely subsided, whereas similar signs did not entirely disappear in Case 9 for a period of fourteen days. Regarding the response of this last patient it should be pointed out that after the first five days of therapy, exhaustion of our supply of ACTH made it necessary to discontinue treatment temporarily during the succeeding five days.

Multiple subcutaneous nodules were a prominent part of the disease picture in 3 patients (Cases 1, 3 and 9). In Case 1 and 9 these lesions were detectably smaller within seven to ten days of the beginning of therapy, and they appeared to be totally absent by the end of six or seven weeks of treatment. In Case 3 the nodules, which were present over the elbows and finger joints, became smaller very gradually and did not entirely disappear until about three weeks after termination of therapy. Furthermore, new nodules appeared shortly afterward on both forearms distal to the olecranon processes.

Only 1 of the patients in this series (Case 8) had chorea. In this case improvement during ACTH therapy was quite striking. Although the hormone was given for a total of only ten days, at the end of this period the chorea, which had been moderately severe prior to treatment, had become quite mild. Thereafter and without further therapy the slight residual chorea gradually lessened and finally disappeared within another three weeks.

Improvement in appetite and gain in weight with ACTH therapy was frequently impressive and was often remarkably prompt. Disappearance of a moderate anemia in association with improvement was likewise quite noticeable in a number of cases.

In several patients who had a marked sinus tachycardia there was considerable slowing of the heart rate soon after ACTH treatment was begun. In addition the hormone may also have played a role in the improvement of 1 patient with paroxysmal auricular tachycardia.*

TABLE 1. *Interval after Beginning of ACTH Required for First Subsidence of Manifestations of Rheumatic Fever.**

CASE NO.	INTERVAL BEFORE MANIFESTATION DISAPPEARED†							
	FEVER	ARTHRALGIA	OBJECTIVE ARTHRITIS	PERICARDIAL FRICTION RUB	ELEVATED SEDIMENTATION RATE	NODULES	CONGESTIVE FAILURE	CHOREA
	days	*days*	*days*	*days*	*days*	*days*	*days*	*days*
1	1	1	—	3	17	45	—	—
2	1	1	—	—	22	—	14	—
3	—	—	—	—	—	‡	§	—
4	1	1	2	—	20	—	—	—
5	2	—	—	11	87	—	56	—
6	1	2	2	—	15	—	—	—
7	1	1	1	1	34	—	—	—
8	—	—	—	—	6	—	—	30
9	1	4	—	14	17	50	—	—
10	1	1	3	—	8	—	15	—

*Figures in the table refer to number of days required for initial disappearance. Certain of these manifestations reappeared later.
†Blanks indicate that manifestation was not present when ACTH therapy was begun.
‡Nodules still present.
§Slight congestive failure possibly still present.

*This patient's case report has not been included in the body of this article because she was treated with ACTH for only four days. She was a thirteen-year-old girl with rheumatic heart disease who was admitted to the House of the Good Samaritan on August 8, 1949, six weeks after the onset of acute rheumatic fever. Paroxysmal tachycardia had been occurring at frequent intervals almost since the onset of her illness and was especially bothersome during the first ten days after admission to the House of the Good Samaritan. At the time ACTH therapy was begun on August 18, the patient was having one of her usual episodes of tachycardia. The sedimentation rate was also elevated. On the second day of ACTH, which was given in daily doses of 70 mg. on the first day and 40 mg. during the next three days, the tachycardia ceased. The sedimentation rate reached normal levels within two weeks, and during a follow-up period of about eleven weeks there have been no further attacks of tachycardia.

The influence of ACTH on rheumatic patients who are in congestive heart failure is especially in need of careful consideration. From our limited experience (4 cases) it appears that the hormone has two opposing actions. Owing either to a direct antidiuretic effect of contaminating posterior-pituitary-lobe principles in the ACTH preparation or to an indirect sodium retaining effect, or to both, the administration of ACTH tends to cause accumulation of fluids and hence aggravation of already existing congestive failure or precipitation of this condition in patients whose hearts are badly damaged. On the other hand, suppression of the active rheumatic inflammatory process and consequent improvement in the myocardium tends to increase renal circulation and excretion and thus to relieve congestive failure. The net result depends upon which of these two effects is the more pronounced and the more immediate.

Our 4 patients (Cases 2, 3, 5 and 10) illustrate both these effects. The patient in Case 2, who was desperately ill and in severe congestive failure, improved with ACTH therapy to such an extent that mercurial diuretics, which she had been receiving frequently for about six weeks prior to hormone treatment, could be entirely omitted after three weeks of ACTH without recurrence of any signs of congestion. In this case it seems likely that the disappearance of heart failure was due to the beneficial effect of treatment on the heart muscle. On the other hand, that therapy was also tending to cause retention of fluids is indicated by the marked diuresis that occurred when it was necessary to discontinue ACTH for a period of three days about one week after this form of treatment had been started.

In Cases 3 and 5, congestive failure, which had been present prior to treatment, became worse during the first few days of ACTH therapy, and did not improve until diuretics were also administered. In these cases the fluid-retaining effects of ACTH apparently outweighed any beneficial effect that the hormone treatment may have had on the heart.

Although congestive failure increased in Case 10, a patient with auricular fibrillation, the fact that this change did not become evident until ten days after ACTH was begun and two weeks after admission to the House of the Good Samaritan suggests that the rapid ventricular rate consequent to lack of digitalis played an important role. Confirmatory of this interpretation is the rapid improvement in the patient's symptoms when he was redigitalized. In any event, it is clear that digitalis was able to control congestive failure after two weeks of ACTH therapy whereas prior to this treatment the patient had a markedly congested liver in spite of full digitalization.

The final rheumatic manifestation to be considered in relation to the assessment of ACTH therapy is the auscultatory signs of heart disease. In conjunction with the subsidence of active rheumatic fever, these signs regressed in 3 patients (Cases 1, 4 and 7).

When the pericardial friction rub disappeared in Case 1, aortic diastolic and mitral mid-diastolic murmurs of moderate intensity were readily heard. As the patient improved, these murmurs gradually lessened in intensity and finally disappeared, the mitral diastolic within twenty-four days and the aortic diastolic in about ten weeks after the beginning of treatment. The changes that took place in the auscultatory findings in Case 7 were similar. A moderately loud mitral systolic and a definite mid-diastolic murmur disappeared about three weeks after ACTH was first given. The patient in Case 4 had been observed by us on numerous occasions prior to her most recent attack of rheumatic fever and was known to have had a moderately loud mitral systolic murmur but no diastolic murmur. With the rheumatic recurrence a definite mitral mid-diastolic murmur developed in addition to the systolic murmur but again disappeared when the patient recovered after ACTH treatment.

In the past, and prior to the use of ACTH at the House of the Good Samaritan, the so-called signs of valvular disease have occasionally been observed to regress and even to disappear spontaneously in association with recovery from active rheumatic fever. The auscultatory finding that disappeared most commonly and most rapidly was the mitral mid-diastolic murmur, which, in early cases of the disease, has been shown not to be due to actual stenosis of the mitral valve.[6] A possible explanation for the production of this murmur in active rheumatic fever has been discussed in a previous publication.[7] Complete disappearance of obviously significant mitral systolic murmurs has also occurred spontaneously but perhaps not quite so frequently, whereas well established aortic diastolic murmurs have only rarely regressed spontaneously to the point of disappearance.*

Therefore, the regression of cardiac murmurs per se is not unusual. Nevertheless, the findings in the patients treated with ACTH are impressive because of the relative rapidity with which the murmurs disappeared and because the auscultatory signs included a distinct aortic diastolic murmur, which, as mentioned above, was observed in the past to disappear quite infrequently.

Bland, White and Jones,[8] in every one of 20 patients with rheumatic fever who died within six months after the onset of their illness, observed a verrucose endocarditis of the mitral valve, but, because there was no stenosis or other deformity of the valve, they believed that the murmurs present during the early months of acute rheumatic fever were probably due in large part to cardiac dilata-

*Bland, Jones and White,[6] in a study of 1000 patients with rheumatic heart disease, reported that among 83 in whom signs of heart disease disappeared there were only 4 cases of an aortic diastolic murmur.

tion secondary to myocardial rheumatism. On the other hand, in the report[6] referred to previously, they considered the persistence of characteristic murmurs after the subsidence of the active phase of rheumatic fever to constitute the most reliable indication of residual valvular injury and expressed the opinion that most of a group of 83 patients in whom they observed murmurs to disappear had injury to the valve cusps at the time of rheumatic fever. It was thought that in these cases the injury was of a very slight degree so that after the subsidence of active rheumatic fever the residual scarring produced no significant deformity or characteristic murmurs.

If the disappearance of definitely significant murmurs in the patients treated with ACTH is interpreted in the light of the experience of Bland and his associates, it seems likely that the rapid changes in the auscultatory findings observed by us were due to a subsidence of the rheumatic inflammatory process at least in the myocardium and possibly in the valves as well. Follow-up observations extending for a number of years will be required before it can be determined whether the subsequent course of patients treated with ACTH who recover without residual signs of valvular disease will be any different from that of patients who previously recovered without the benefit of hormone therapy. In the latter group, in the absence of recognized recurrent rheumatic fever, it has been reported that within ten years signs of heart disease developed insidiously in about 8 per cent of patients.[9]

In connection with this discussion it should be pointed out that from our knowledge of the pathology of rheumatic heart disease one would not expect long-standing valvular deformity to improve with ACTH, or for that matter with any other form of medical treatment known to us at present. Nevertheless, the observations in our 3 cases indicate the promise that ACTH holds of being an effective therapeutic agent for the prevention of heart disease when used in the early stages of rheumatic fever and also for preventing progression of valvular damage in cases of previously established rheumatic heart disease.

Withdrawal Effects

The course of these patients, when the dosage of the hormone was reduced and when therapy was entirely omitted, is of considerable importance in relation to the problems of determining the optimum therapeutic schedule of ACTH and of determining whether or not an attack of rheumatic fever can really be shortened by this form of treatment. For the sake of brevity only episodes that seemed definitely to be related to change in dosage of ACTH are reviewed. A few cases of fever in which other factors may have played a role are omitted from the discussion.

The clinical course in Case 1 was one of steady improvement, but this was not entirely true of the course of the sedimentation rate, which fell within a period of sixteen days from an initially extremely high level of 1.7 mm. to a normal rate of 0.28 mm. per minute and thereafter remained well within normal limits for an additional two weeks. During this time ACTH had been reduced from an original dose of 9 mg. four times daily (total of 36 mg. per day) to 5 mg. four times daily (total of 20 mg. per day). Four days after the hormone was further reduced to 3 mg. four times daily (total of 12 mg. per day) the sedimentation rate rose and remained at a slightly elevated level (0.45 to 0.65 mm. per minute). However, even though ACTH was further reduced and finally omitted altogether, the sedimentation rate again came down and thereafter remained well within normal limits.

In Case 2, when after one week of treatment exhaustion of our supply of ACTH necessitated omission of the hormone for a period of three days, there was a prompt rise in the temperature to 102°F. and an increase in the level of the sedimentation rate as well as a recurrence of chest and back pain. Subsequently, after the patient had again been responding well to treatment, the sedimentation rate rose from a normal to a slightly elevated level a few days after the dose of ACTH was reduced from 5 mg. four times daily (total of 20 mg. per day) to 3 mg. four times per day (total of 12 mg. per day). When a week later the hormone therapy was terminated, there was a transient cycle of low-grade fever, and the sedimentation rate rose steadily to 1.8 mm. per minute. Thereafter, although no further treatment was given, the temperature remained normal, and the sedimentation rate settled down to the moderately elevated level of between 0.60 and 0.80 mm. per minute.

In Case 4 the fever and arthritis responded promptly to ACTH and the sedimentation rate dropped from a pretreatment level of about 1.5 mm. per minute to 0.42 mm. within three weeks, during which the hormone was reduced from 7.5 mg. four times daily (total of 30 mg. per day) to 3 mg. four times daily (total of 12 mg. per day). Further reduction in the dose to 1.5 mg. four times daily (total of 6 mg. per day) was followed shortly by a rheumatic exacerbation characterized by fever, joint pain and swelling, and elevation of the sedimentation rate to 1.6 mm. per minute. It is of interest that in spite of a continuation of the same low dosage schedule this rheumatic cycle again subsided within about seven days, and, even though the hormone was completely omitted after another two weeks, improvement continued without any further cyclic increases in rheumatic activity.

The patient in Case 5 was a very ill girl with fever, pericarditis and congestive failure. During the first ten days of ACTH treatment, when the total daily dose was at first 50 mg. and then 30 mg., she showed

slow but nevertheless definite improvement especially after mercurial diuretics were used to help combat the congestive failure. On the twelfth day of treatment the dose of ACTH was reduced to 3 mg. four times daily (total of 12 mg. per day) and one week later, while this dosage schedule was still being maintained, there was a four-day cycle of low-grade fever and a rise in the sedimentation rate. This episode again began to subside, and a little later the treatment schedule was changed to 3.3 mg. three times daily (total of 10 mg. per day). However, about two weeks after the previous cyclic increase in rheumatic activity the patient again became worse, with fever, rise in the sedimentation rate and signs of pericarditis. In association with an increase in the dose of ACTH to 5 mg. three times daily (total of 15 mg. per day) this episode promptly subsided. This same dosage was then continued for two months, during which the patient steadily improved and became free from all signs of rheumatic fever. When ACTH was finally omitted, there was no further evidence of increased rheumatic activity.

In Case 9, high fever responded promptly to ACTH, but, when after five days of treatment exhaustion of our supply made it necessary to substitute saline injections for the hormone during a five-day period, there was an immediate febrile response, the temperature reaching a maximum of 103°F. on the fourth day after omission of ACTH.

In summary, 3 of 6 cases of ACTH-withdrawal reactions promptly responded to resumption of therapy or increase in dosage. It is of interest that 3 other episodes associated with reduction in dosage or omission of therapy spontaneously subsided within a relatively short period even though the treatment was not altered.

Relation of Response to Dosage and Duration of Therapy

All our patients were treated at the beginning by the administration of ACTH at six-hour intervals. In most cases this schedule continued until close to the end of the treatment period. An attempt was also made in most cases to reduce the total daily amount of ACTH slowly, and in some cases this was partly effected by a final reduction in the frequency of injections to eight-hour and even to twelve-hour intervals.

Initial individual doses of 7.5 to 12.5 mg. and total daily amounts of 30 to 50 mg. appeared in most cases to be sufficient to cause prompt clinical improvement. Such doses usually caused a significant depression of the total circulating eosinophils, a change that indicates a good response of the adrenal cortex to the ACTH.[5]

Although of the 8 patients in whom therapy has been terminated 1 (Case 5) was treated for as long as fourteen weeks and others (Cases 1, 2, 3, 4) for between five and seven weeks, 2 patients (Cases 6 and 7) showed an excellent response to a total of four weeks of ACTH, and 1 patient with chorea (Case 8) apparently reached a state of complete quiescence shortly after a treatment period of only ten days. Furthermore, the previously discussed clinical withdrawal reactions to reduction in ACTH below certain critical levels, as well as changes in total circulating eosinophils, suggest that in the latter portions of the treatment periods of some of the patients subeffective doses of the hormone were being administered. The clinical withdrawal reactions that were observed usually occurred when the individual dose of ACTH was reduced below 5 mg. and when the total daily amount fell below 15 or 20 mg. As a rule there was a significant increase in the number of circulating eosinophils with similar changes in dosage.

Although these data suggest that most patients with rheumatic fever will require a minimum individual dose of 5 to 10 mg. and a minimum total daily dose of between 20 and 50 mg., it should be pointed out that 2 patients (Cases 6 and 7) who showed a striking therapeutic response received doses of this size for only four days, whereas another patient (Case 3) failed to show any clear-cut response in spite of the fact that she received this amount of ACTH during her first two weeks of treatment. Obviously, there must be considerable individual variation in the amount of ACTH and duration of treatment necessary for an optimum therapeutic effect on the disease. Further study of many cases will be necessary to determine the limits of these variations and to determine whether patients will be encountered who will be refractory to even very large doses of ACTH.

Untoward Reactions

The tendency of ACTH to cause retention of fluids and thus to increase previously existing congestive failure or to precipitate this condition in patients with severe rheumatic carditis has already been discussed. However, this risk does not seem to constitute a valid contraindication to the use of therapy, because such congestive failure, at least in our limited experience, can be adequately controlled by salt restriction and the proper use of diuretics. Furthermore, suppression of active carditis by ACTH in the long run is beneficial to the myocardium and eventually may bring about improvement in congestive failure.

The only other serious untoward reaction from ACTH encountered in our 10 patients was the severe mental depression that developed in Case 2 after treatment for about one month. During the first week after omission of the hormone this condition became even worse but then gradually improved over a period of about a month. This patient, early in treatment, suffered from severe abdominal cramps, which apparently were due to a high Pituitrin content of the hormone preparation.

When another batch of ACTH was used, the difficulty was no longer encountered.

Other relatively minor side effects from ACTH were transient headaches in 2 cases, slight striae in 2 cases, acne in 6 cases and development of varying degrees of roundness of the face in 6 cases. The last two manifestations usually became apparent during the second to fourth week of treatment and lessened gradually after discontinuation of therapy.

Elevation of the blood sugar, glycosuria and hypertension have not been observed in any of the 10 patients. However, in connection with this discussion, it should be pointed out that in general we have used relatively small doses of ACTH in comparison to the doses used by others in patients in whom such complications have been reported.

Mechanism of ACTH Action in Rheumatic Fever

The evidence at hand suggests that the effect of ACTH in rheumatic fever is indirect and depends upon stimulation of the adrenal cortex and a consequent increased production of 11, 17 - oxysteroids.[3] However, the way in which the adrenocortical steroids suppress the inflammatory rheumatic process is still not understood.

Whether the various effects that certain steroids have been observed to have on sodium, potassium, carbohydrate and protein metabolism have anything to do with the beneficial influence of Cortisone and ACTH in rheumatic fever is entirely speculative. Administration of ACTH causes an increased urinary excretion of uric acid, a transient decrease in circulating lymphocytes, a somewhat more prolonged decrease in circulating eosinophils[3] and, at least in patients with active rheumatic fever, a rather marked reduction in the serum concentration of gamma globulin and in the titer of antistreptolysin O.[10] Whether any of these changes are at all related to the basic physiologic disturbance in rheumatic fever is not known.

The effect of ACTH on the circulating eosinophils is of especial interest in view of the hypersensitivity hypothesis of rheumatic fever and the known relation of eosinophilia to allergic states. Of more practical importance is the fact that the decrease in circulating eosinophils serves as a rough measure of adrenocortical response to ACTH. Thus, a failure to obtain a significant reduction in circulating eosinophils suggests either that the amount of ACTH being administered is inadequate or that the adrenal gland is unresponsive. Rheumatic-fever patients with adrenocortical insufficiency due to primary disease of the adrenal gland or physiologic exhaustion should show a very poor eosinophil response[5] and would not be expected to be benefited by ACTH therapy. However, in this connection, it is worthy of note that 2 of our patients (Cases 6 and 7) who improved rapidly with relatively small doses of ACTH failed to show an appreciable decrease in circulating eosinophils.

Finally, although the response of patients to ACTH therapy seems to suggest that an underlying endocrine defect is involved in the mechanism of rheumatic fever, there is as yet no evidence to substantiate this inference, and the possibility that suppression of the rheumatic inflammatory process by changes effected through hormone administration could be fortuitous cannot be dismissed.

Summary and Conclusions

ACTH was administered for periods of ten days to fourteen weeks to 10 patients with active rheumatic fever.

Most of the important manifestations of rheumatic fever were represented by these 10 patients. The majority of the patients were severely ill and suffered from a high degree of active carditis.

In general, the clinical response was satisfactory in all but 1 of the 10 patients, but the speed and extent of the response varied considerably. In the light of previous experience at the House of the Good Samaritan the improvement shown by the majority of the patients was quite striking.

In spite of transient withdrawal reactions encountered in some cases at the end of therapy, the rheumatic process finally subsided within four to ten weeks in 5 of 8 patients who were followed for periods of one to four months after the completion of treatment. In 1 of the remaining 3 patients the response was slower although a state of quiescence was finally attained. Another very ill patient improved considerably but still has low-grade, active rheumatic fever. In the third patient there is no definite evidence that ACTH was responsible for any improvement.

Definitely significant systolic or diastolic murmurs, or both, regressed in 3 patients, and in 2 of these (both early cases) the auscultatory signs completely disappeared within three to ten weeks.

ACTH therapy seems to have two opposing actions in patients with active rheumatic carditis and congestive failure. A tendency of the hormone to cause retention of fluids may result in an aggravation of the latter condition. On the other hand, suppression of the active rheumatic process and consequent improvement in the myocardium tends in the long run to relieve congestion. In cases in which ACTH caused an accumulation of fluids, congestive failure could be controlled by a low-sodium diet and mercurial diuretics.

In addition to increase in previously existing congestive failure the only other important untoward reaction to ACTH therapy was the development of severe mental depression in 1 patient. Minor side effects have been abdominal cramps, headache and the development of roundness of the face, acne and skin striae.

Although no final conclusions regarding optimum dosage of ACTH can be drawn from this small series of cases, it appears that the minimal effective

individual dose may be between 5 and 10 mg. and the minimum daily dose between 20 and 50 mg.

ACTH probably acts by causing an increased endogenous production of 11, 17-oxysteroids by the adrenal cortex, but the mechanism by which the adrenocortical steroids in turn suppress the active rheumatic process is not known.

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TYPHOID ENTEROCOLITIS SIMULATING CHRONIC BACILLARY DYSENTERY

Report of a Case with Cure by Chloromycetin

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THE current literature is replete with accounts[1-4] of typhoid fever occurring in patients who had previously received prophylactic inoculations. Furthermore, there are numerous reports dealing with asymptomatic carriers and the wide variety of therapeutic measures, both medical[5-10] and surgical, designed to eradicate the organism. The case of chronic typhoid infection of the intestinal tract reported below presents several unique features: chronic dysentery of four years' duration due to *Eberthella typhosa* in a man who had been previously immunized against typhoid; absence of any antecedent febrile illness suggestive of typhoid fever; proctosigmoidoscopic evidence of chronic colitis and x-ray changes involving the small intestine; and rapid reversal to normal of symptoms, sigmoidoscopic, roentgenographic and bacteriologic findings after large doses of Chloromycetin.

As far as we are able to determine this is the first report of a case of chronic intestinal infection simulating chronic dysentery caused by *E. typhosa*.

Case Report

L. G., a 38-year-old salesman, was referred for investigation on July 5, 1949, because of recurrent bloody diarrhea of 4 years' duration. The patient stated that he had been perfectly well prior to his induction into the Army in March, 1942, and had remained so until the latter part of 1945, when, while he was in Honolulu, mild "dysentery" developed, for which he received only symptomatic treatment. He never experienced any febrile illness and was never hospitalized during his Army career. He stated that he had received "booster" injections of typhoid, paratyphoid A and paratyphoid B every 18 months after his initial inoculation upon induction and believed that the last one had been administered early in 1945.

After the initial attack of "dysentery," he noted recurrences of diarrhea and abdominal cramps every 2 or 3 weeks, lasting for 4 or 5 days and accompanied by blood in the stool. These symptoms continued when he returned to civilian life in March, 1946, and persisted to the present. In addition he complained of intermittent stabbing pain in the right upper quadrant radiating along the costal border, unrelated to meals. There were frequent heartburn, gaseous distention and poorly localized abdominal cramps, but no qualitative dyspepsia. His appetite was capricious, at times being normal but followed by periods of anorexia corresponding to days when his abdominal discomfort and diarrhea were pronounced. He had lost 15 pounds since the onset of symptoms and was unable to regain the lost weight. He noted easy fatigue and frequent headaches, which he related to his "poor digestion," but during brief remissions all his symptoms disappeared.

He had been investigated on two occasions by sigmoidoscopy and barium enema and had been told that he had a spastic colon with bleeding hemorrhoids and that his symptoms were of nervous origin. He had been placed on a bland, low-residue diet, with Metamucil, sedatives and antispasmodics, but had experienced no relief with any form of therapy.

Physical examination revealed a moderately well nourished adult male who did not appear ill. The temperature, pulse and respiration were normal. Positive physical findings were limited to the abdomen, where there was mild generalized tenderness most marked in both lower quadrants. Neither the liver nor the spleen was palpable.

Proctosigmoidoscopic examination disclosed irregular areas of granular mucosa distributed in patchy fashion in the lower rectum and in the sigmoid, the intervening mucosa being normal. The submucosal lymph follicles were prominent in the involved areas, and the mucosa was unusually friable, bleeding on slight touch. No ulcerations were seen, and there were no hemorrhoids. The gross appearance suggested a proctosigmoiditis of infectious origin such as is seen in shigella or salmonella infection. The changes were not the diffuse type characteristic of idiopathic ulcerative colitis. Direct smears showed a highly granular exudate with no amebas. Culture‡ of material taken by swabs from the sigmoidal lesions on July 5 revealed *E. typhosa*.§ This examination was repeated on July 7 and 8, with the same findings, verified by the New York City Board of Health.

The urine was normal. Examination of the blood showed a red-cell count of 4,520,000, with a hemoglobin of 14.5 gm., with a white-cell count of 8200, 72 per cent polymorphonuclear leukocytes (6 per cent stab forms, 65 per cent neutrophils and 1 per cent basophils), 22 per cent lymphocytes and 6 per cent monocytes. The sedimentation rate was 18 mm. in 1 hour (Cutler method). Warm stools following administration of magnesium sulfate were negative for ameba (4 specimens). Culture of the urine and bile showed no pathogens.

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‡Material from swabs was implanted directly into Selenite F broth, then transferred to eosin-methylene blue, shigella-salmonella, and Wilson-Blair agar and finally transferred to Kliegler's iron agar.

§Culture of the stools of the patient's wife and three-year-old child were negative for *E. typhosa*.

The New York City Board of Health reported negative serum agglutination for typhoid, paratyphoid A and B, and *Proteus OX19* and 1:20 positive reaction for *Brucella abortus*. On roentgenographic examination the gall bladder, stomach and duodenum were normal. Serial x-ray films of the small intestine revealed marked segmentation and puddling involving the entire ileum, with loss of normal mucosal markings (Fig. 1). Some of the loops were dilated, and there was marked hypermotility of the intestine, barium reaching the cecum after 1 hour. Barium enema disclosed feathering of the margins of the sigmoid, suggesting mucosal ulcerations in this region, but the postevacuation films of this region were normal.

The patient was hospitalized on July 14, and Chloromycetin was administered orally in doses of 1.0 gm. every 3 hours (8 gm. daily) for 7 days, followed by 1.0 gm. every 4 hours (6 gm. daily) for 7 days. He received a total of 98 gm. of Chloromycetin in 14 days. On the 5th day he complained of severe burning of the tongue and pharynx so that the ingestion of all foods was painful. There was severe heartburn and an increase of diarrhea accompanied by rectal burning, which persisted during the entire period of medication. All these symptoms, as well as the intense hyperemia of the entire buccal mucosa, disappeared within 3 days of the discontinuance of the drug. One week later all abdominal discomfort had disappeared. Sigmoidoscopy on August 8 showed an entirely normal mucosa, and repeated subsequent examinations have been normal. Culture of stools and rectal swabs have been repeatedly normal over a 5-month period. Barium enema on October 1 was normal. Serial x-ray films of the small intestine on October 3 showed that both the motility and mucosal pattern had reverted to normal, although there was slight residual segmentation. Culture of the bile on November 21 was negative. Two diarrheal stool specimens obtained after instillation of magnesium sulfate through the duodenal tube during biliary drainage were negative on culture. The patient has been completely asymptomatic throughout the 5 months following therapy. He reports that his appetite is normal and that he can perform his work without fatigue for the first time since the onset of his illness in 1945.*

Discussion

Although the low incidence of typhoid resulting from the use of vaccine has been one of the major feats of preventive medicine, it is well known that immunization is not absolute. The disease may occur regardless of how strong an artificial immunity has been produced. The prophylactic action of the vaccine lasts for five or six months, after which immunity is lowered. Most observers who have studied the disease occurring in persons who had been vaccinated agree that it presents an atypical clinical picture, with an insidious onset, and is frequently difficult to diagnose. It seems likely that, in the case reported above, a form of typhoid developed in 1945 and was rendered extremely mild by previous immunization, and in the absence of fever the condition was considered to be a mild "dysentery" or "food poisoning."

The typhoid-carrier state may result either with or without previous active disease. Thus, Saphir, Baer and Plotke,[5] in their report on 110 typhoid carriers, found that 56 (50.9 per cent) gave no history of previous typhoid fever. As is generally true of carriers, their patients felt remarkably well and had no symptoms suggestive of chronic infection. The patient discussed above, however, can hardly be termed an asymptomatic carrier since he had an active enterocolitis with intermittent symptoms referable to the intestinal tract for five years. Although this syndrome is not uncommon after bacillary dysentery due to shigella or salmonella, it is apparently extremely rare after that due to *E. typhosa*. The view has been advanced[11] that chronic ulcerative colitis is the end result of previous bacterial infection. In not a single case, however, has *E. typhosa* been implicated in this transition.

It must be emphasized that the sigmoidoscopic changes, with patchy distribution, in no way suggested idiopathic ulcerative colitis. Furthermore, the abnormality of the small intestine demonstrated by x-ray examination did not resemble that seen in idiopathic ileocolitis. This case appears to be similar to 3 cases reported by Silverman and Leslie[12] in which chronic dysentery was caused by *Salmonella schottmülleri*. The significant features in their cases were sigmoidoscopic signs of inflammation without ulceration and negative x-ray examination of the colon, but roentgenographic changes in the small intestine characterized by flocculation of the barium and abnormal segmentation, the so-called deficiency pattern. This pattern was quite pronounced in the case reported above, but whether

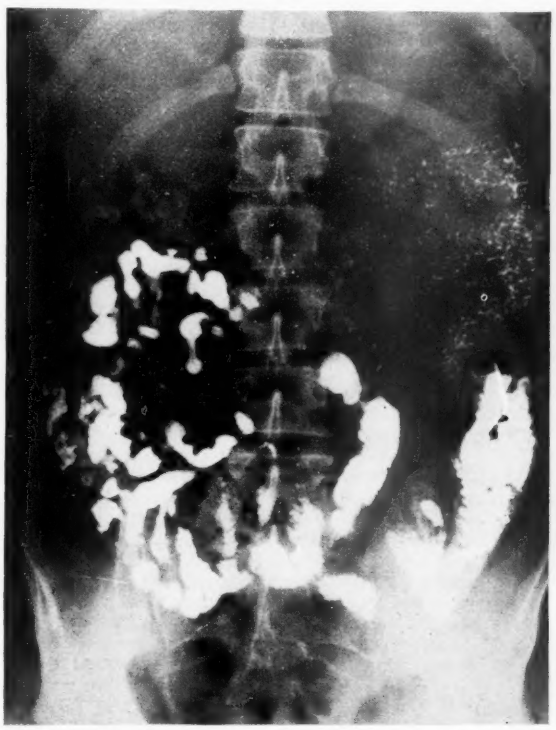

Figure 1. *Film Taken One Hour after the Ingestion of Barium (July 9, 1949), Showing Marked Segmentation of the Entire Ileum, with Puddling of Barium in the Loops, Which Show a Loss of Normal Mucosal Pattern.*

There is considerable hypermotility, since the barium has reached the cecum whereas the stomach has no barium residue.

*After this paper was submitted for publication (9 months after therapy) examination showed an asymptomatic patient with negative stool cultures.

it was due to inflammatory changes or nutritional disturbance or both could not be determined.

The absence of agglutinins against typhoid as exhibited by the negative Widal test is interesting but not unusual since this has been noted even in cases of typhoid fever,[13] as well as in carriers.[5]

Collins and Finland[10] have indicated that Chloromycetin is not completely effective as a curative agent in typhoid fever, and that it was ineffective in eradicating the organism from a typhoid carrier in whom a cholecystectomy had previously been performed. Similarly, relapses in the treatment of the acute disease were observed by Woodward and his co-workers.[14] Collins and Finland have suggested that response to Chloromycetin may vary with differences in strains of the organism or with the nature of the lesion. The excellent results obtained in the case reported above may be explained in part by the large doses of Chloromycetin employed, or possibly by the fact that the disease was limited to the intestinal tract without biliary-tract involvement.

Summary

A case of typhoid enterocolitis simulating chronic bacillary dysentery is presented.

The patient had been inoculated against typhoid prior to the onset of diarrhea and never exhibited signs of typhoid fever.

Proctosigmoidoscopic evidence of inflammation was present, as well as marked abnormalities in the small intestinal pattern.

A complete symptomatic cure as well as a rapid reversal of bacteriologic, sigmoidoscopic and x-ray findings was obtained after a two-week course of Chloromycetin (the total dose was 96 gm.).

All findings have remained normal during a five-month follow-up period of observation.

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RECURRENT POST-INFARCTIONAL SHOULDER-HAND SYNDROME*

Report of a Case with Unusual Clinical Evolution

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THE shoulder-hand syndrome may be defined as a combination of painful shoulder disability with homolateral pain and swelling of the hand. This entity has recently been described in detail by Steinbrocker and his associates[1] in an extensive report and review of the literature. The causes include: peripheral lesions such as trauma and suppuration of the extremity (Sudeck's atrophy, causalgia and so forth), vascular disease (thrombophlebitis, periarteritis nodosa), cervical osteoarthritis and post-infarctional heart disease; lesions of the spinal cord and ganglions such as herpes zoster; cerebral lesions (hemorrhage, thrombosis) and a large number classified as idiopathic.

The syndrome has been divided into three phases. The first is manifested by pain, limitation of motion and diffuse tenderness of the shoulder, with swelling, pain and stiffness of the hand and fingers. This is followed by a second phase in which there is gradual relief of the shoulder pain and dysfunction with diminution of the swelling of the hand. Stiffness and flexion deformity of the fingers become more pronounced, and there is atrophy of the subcutaneous tissues and intrinsic muscles as well as trophic skin changes. X-ray examination reveals patchy osteoporosis of the bones of the hands. In the third phase there are marked trophic changes of the hands.

During the past year we have observed a patient with postinfarctional bilateral shoulder-hand syndrome with an unusual clinical evolution. This case was as follows:

CASE REPORT

M. L., a 64-year-old man, entered the hospital on May 3, 1948, because of precordial pain of 4 hours' duration, radiating down both arms. For several months he had experienced many episodes of substernal distress on exertion, which were relieved by nitroglycerin. These attacks had increased in severity and frequency in the 2 weeks before entry. However, on the day of admission 15 tablets taken consecutively had given no relief. The past history was otherwise not contributory.

Physical examination showed moderate cyanosis. The cervical veins were not distended. The heart was enlarged, the border of cardiac dullness extending 2 cm. to the left of the midclavicular line in the fifth intercostal space. Numerous premature beats were heard, but no murmurs were detected.

The blood pressure was 152/80. The temperature was 98°F., the pulse 112, and the respirations 42.

Electrocardiograms showed wandering of the pacemaker within the sinoauricular node, numerous multifocal premature ventricular systoles, and changes diagnostic of a recent anterior-wall infarction. The white-cell count was 14,500. There was a ++ test for albumin. The blood Kahn reaction was negative, and the nonprotein nitrogen was 40 mg. per 100 cc.

The patient received oxygen, morphine, quinidine and papaverine. During the 5th week of hospitalization he complained of pain in the shoulders and hands. No abnormal physical findings were observed. A roentgenogram of the chest revealed cardiac enlargement, with prominence of the aortic knob. He was discharged, greatly improved, 52 days after admission.

He returned to the Cardiac Clinic 11 days later complaining of severe pain in both shoulders and pain and swelling in both hands. These complaints prevented him from sleeping or engaging in minimal activity. There were no cardiovascular symptoms. Bilateral stellate-ganglion blocks with 1 per cent procaine were performed, with no improvement.

The patient re-entered the hospital 6 weeks later because of continued shoulder and hand pain, which had not been relieved by salicylates, acetophenetidin or codeine. Roentgenograms of the cervical vertebras, shoulders and hands showed no bony abnormalities. On two occasions bilateral cervicothoracic sympathetic blocks extending from the eighth cervical to the third dorsal segment and, on four occasions, stellate-ganglion injections were performed, with no relief of pain or objective improvement of the hands. He was discharged 22 days after the second entry.

He was readmitted to the hospital 6 weeks later because of the simultaneous onset of acute precordial pain with severe pain and swelling of the hands, awakening him from sleep. On admission the blood pressure was 140/80, the respirations were 38, the pulse 120, and the temperature 99°F. Marked cyanosis was present. The heart tones were indistinct, with a grossly irregular rhythm. There was no other evidence of circulatory failure. The hands were extremely swollen, tender and reddened, with atrophy of the overlying skin. The white-cell count was 14,150, with 95 per cent neutrophils. The sedimentation rate was 32 mm. in 1 hour. The initial electrocardiograms revealed transitory auricular fibrillation. Although the subsequent electrocardiograms were not entirely typical, the clinical attack and evolution were characteristic of a repeated myocardial infarction. The patient was discharged after 6 weeks, with persistent shoulder and hand symptoms.

He was readmitted 9 days later because of continued pain in the shoulders and hands. Roentgenograms of the shoulder and cervical vertebras were again normal, but minimal osteoporosis was noted in the hands. Physiotherapy gave little relief, and he was discharged on January 15, 1949.

The final admission occurred on May 9, because of the onset of sudden precordial pain, dyspnea and orthopnea of 3 hours' duration. The blood pressure was 110/70; the pulse was 140, the respiration 34, and the temperature 99°F. The heart tones were regular but faint. There were moist inspiratory rales at both lung bases. The liver edge was palpated 6 cm. below the costal margin. There was slight pitting edema of the legs. An electrocardiogram revealed a recent posterior myocardial infarct. The patient died 2 days later. Permission for autopsy was not obtained.

DISCUSSION

Prior to the second myocardial infarction at the third admission, the swelling of the hands had subsided almost entirely, with residual stiffness and pronounced flexion deformity of the fingers (Phase II). This attack was manifested by the sudden onset of precordial pain and the simultaneous recurrence of severe pain in both shoulders, with pronounced swelling and redness of the hands and fingers (Phase

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I). The changes in the hands were so prominent that the patient's wife volunteered the observation.

On reviewing the literature we have found no other case in which there was recurrence of Phase I after progression to a later stage had taken place.[2-9] This recurrence is important because it provides additional evidence that the myocardial infarct was a precipitating event for the changes in the shoulders and hands.

Theoretically, the changes in the upper extremities result from a neurovascular reflex mediated through the internuncial pool of the spinal cord.[10] This vicious circle is initiated and maintained by impulses arising in the region of the primary pathologic process. We believe that the prompt recurrence of Phase I at the onset of the second myocardial infarct may be explained by impulses set up in this area utilizing the previous pathway in the internuncial pool initiated by the first infarct.

This case clearly illustrates that the severe symptoms and disabling progression of the reflex dystrophy may overshadow the original cause in therapeutic importance. From the onset of the pain in the shoulders and hands until his death approximately one year later, the patient was unable to sleep or to eat comfortably because of this complaint. Analgesics, opiates and physiotherapy afforded only temporary relief.

Block or excision of the stellate and upper dorsal ganglions has been recommended for the treatment of such reflex dystrophic states.[1, 11, 12] These procedures could be expected to be effective by interrupting a limb in the sympathetic reflex arc and thus breaking the vicious circle. Although in the case reported above several stellate and upper dorsal blocks were not helpful, failure could be ascribed to the fact that this was not tried before Phase II appeared.

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MEDICAL PROGRESS

NEUROSURGERY, 1941-1950 (Concluded)*

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BOSTON

Surgical Intracranial Infection

Brain Abscess

As was to be expected with the advent of the sulfonamide drugs and the antibiotics, interest in the strictly neurosurgical infections of the central nervous system and its coverings has increased greatly in the past nine years. The therapy of brain abscess in particular has had an interesting evolution. In 1941, the premise that the therapeutic possibilities of encapsulated and unencapsulated abscesses could not and should not be the same in detail was generally recognized. It was held that treatment of the first should be by surgical drainage from within the capsule as exemplified by the use of Mosher's wire-gauze drain and that of the second by methods looking toward the localization of a spreading purulent encephalitis within an artificially created capsule as exemplified by King. The encapsulated abscess was considered to be the result of a local spread of infection whereas the unencapsulated encephalitis usually developed as the result of infected emboli coming from a distance and producing among other conditions a spreading infectious thrombosis and septic infarction of the cerebrum.

In 1941 Grant[219] reported the end results in 100 consecutive cases of brain abscess. He concluded that "the initial attack . . . should be carried out through a small trephine opening with the insertion of a drainage tube." His mortality at that time was 53 per cent and covered the years from 1926 to 1940. Most if not all of his patients had encapsulated abscesses. In the same year Grant[220] presented a well documented review of the problem of brain abscess and concluded from the literature that "the best surgical results follow drainage after encapsulation had occurred." He stated that the average mortality would be about 40 per cent. Schreiber[221] came to essentially the same conclusion about cerebellar abscesses of otitic origin. In 1945 Pennybacker,[222] following the principle of repeated tapping with eventual total extirpation of the capsule and its contents as originally advocated by Vincent et al.,[223] reported on 50 cases,

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26 of which were treated in this way. He makes the point that spread of the infection to the ventricular or subarachnoid spaces and increased intracranial pressure are the two factors with which treatment is concerned. He also stresses the importance of treating the original local source of infection (as in otitic and sinus infections) as a necessary early adjunct to the abscess therapy — a point that had previously been insufficiently emphasized. He used Thorotrast as first described by Kahn[224] to visualize the abscess cavity, as well as arteriography and ventriculography for more exact localization, but did not touch upon the problem of the unencapsulated abscess per se except to note that "the patient must be kept alive until the abscess has a wall of sufficient thickness to enable it to be dissected out," which is estimated to take from five to seven weeks.

Furlow[225] discussed the use of penicillin in combination with the sulfonamides parenterally as already advocated in principle by Pennybacker.[222] Glaser and McGregor[226] reported a cure following the use of penicillin in the abscess cavity itself. Fincher[227] described 5 cases in which Pennybacker's method was used after the patient had been fortified against spread of infection with sulfonamides and penicillin parenterally. At the time of operation the abscess was exposed through a craniectomy and collapsed by suction, and the collapsed capsule was dissected free and removed through a small transcortical incision after which the abscess site was lined with gel-foam soaked in penicillin. The wounds were sutured as clean wounds without drainage. All patients recovered. Le Beau and Guiot[228, 229] point out that Thorotrast may not fill all the locules of a multilocular abscess and that hence too much reliance should not be placed on its diagnostic value in the face of a downhill clinical course. Tolosa[230] does not agree with them. They advocate total extirpation, preferably after puncture and aspiration, as well as the use of penicillin in the wound. Wyke,[231] writing of severe intracranial infections, advocates daily intraventricular injection of penicillin through needles inserted into the ventricles by way of trephinations and after irrigation of the ventricles by warm Ringer's solution. He minimizes the possible convulsive effect of the intraventricular injection of penicillin but gives no figures by which one may judge of the validity of his conclusions. Cairns et al.,[232] in discussing complications of head wounds with especial reference to infection, cite 14 cases with chronic brain abscesses. All but 2 were encapsulated even though these 2 patients had been wounded two or three years earlier. Retained bone fragments were present in most cases, and a retained missile in 1. Total extirpation of the capsule and its contents was given as the therapy of choice and was made possible without undue risk of spreading infection by the use of the sulfonamides and penicillin. Pennybacker[233] points out that success had attended his efforts at excision either after emptying the abscess cavity or at his first exposure in 7 patients, all of whom recovered. He ascribes this to the use of penicillin parenterally, intraventricularly and at the site of the removal.

Pennybacker and Sellors[234] approach the problem of metastatic brain abscesses from the point of view of the thoracogenic abscesses. Here, again, they claim that penicillin given if necessary as described above had enabled them to deal successfully with problems that could not be handled in the prepenicillin era. However, they rightly insist, as the senior author had previously done,[222] that even with the brain abscess cured, the primary focus in the lung must also be eliminated if later recurrent abscesses in the brain are to be eliminated. They have even excised with success 2 acute abscesses that were in the stage of encephalitis; this procedure would certainly have caused a generalized intracranial infection in the prepenicillin period and may, for all the evidence to the contrary, still cause such an infection if the infecting agent is not penicillin sensitive. Le Beau[235] shares their faith in this antibiotic and reports on 41 cases of brain abscesses and 9 meningeal abscesses. Twenty-two of the brain abscesses were multilocular; nevertheless, excision, usually preceded by aspiration and always done in conjunction with penicillin by the routes noted above, was practiced in every case. Although the point is not specifically stated it is probable that none of the patients had unencapsulated abscesses by the time they were first seen. His data on meningeal abscesses are discussed below under "subdural empyema." He reports an over-all mortality of 15 per cent. Tutton and Shepherd[236] describe the technic, advantages and disadvantages of "pyograms." These are injections of Thorotrast into brain abscess cavities. The Thorotrast, after an interval of thirty-five to forty-two days, will visualize the capsular wall as being 1 mm. thick when it has been injected six to forty-three days after the estimated start of the abscess. Only after this has happened can change in size and shape of the abscess be visualized serially without further injection of more Thorotrast. It does not fill all the loculi in all multilocular abscesses.

It appears that the advent of the sulfonamides and the antibiotics such as penicillin and streptomycin have now made it possible to control the increased intracranial pressure associated with cerebral and cerebellar abscesses by repeated tapping and emptying by suction of encapsulated abscess cavities without danger of spreading infection generally throughout the intracranial cavity. After the surgeon is satisfied that the capsule is thick enough to withstand traction, he can incise it and its contents *en bloc* and suture the wound as though it were a clean one. Complications may be ex-

pected from missed loculi of multilocular abscesses, recurrent metastatic abscesses if the original extracranial or cranial focus is not removed and from possible necessary treatment prior to encapsulation. Other methods of treating encapsulated abscesses are now outmoded. The treatment of unencapsulated abscess is still *sub judice*. If it cannot be delayed until a capsule has formed and the infection *must* be treated while in the stage of encephalitis total excision by suction is the alternative choice to King's method of endeavoring to stimulate capsule formation by lining and holding open with gauze the cavity of a centrally excised infected area. Thorotrast delineation of abscess cavities, in conjunction with ventriculography and arteriography, is an essential step in the preoperative localization of most cerebral abscesses. These procedures will also be helpful in cerebellar abscesses and in cerebral abscesses whose location is obvious in any case. Thorotrast will not localize an infected area that is not sufficiently encapsulated to permit tapping. It is as important to treat effectively the point of origin of the bacteria that cause an intracranial abscess as it is to treat the abscess itself.

Subdural Empyema, Subdural Abscess, Purulent Pachymeningitis, Meningeal Abscess and Subdural Suppuration

These various synonyms have been used in the literature in reference to infection in the cerebral subdural space. Probably the best terms are subdural suppuration and purulent pachymeningitis as proposed by Schiller, Cairns and Russell.[237] An adequate descriptive name for the condition under discussion is not without importance. For example, subdural empyema as described by Kubik and Adams[238] and Courville[239] is a rapidly advancing, nonlimited, purulent, extremely fatal infection of a part or the whole of the cerebral subdural space. It commonly results from the spread of infection from the ear or nasal sinuses and is accompanied by venous thrombosis and surface destruction of the cortex underlying the infected portion of the subdural space. Subdural abscess is a much more benign form of the same condition.[240] Here the process is limited to a part of the subdural space. It advances slowly, may be present for years and may be the end stage of the more virulent but nonfatal treated or untreated forms of subdural empyema. It may follow trauma, otologic and sinus infections or may develop as the result of septic emboli from distant sources. The collections of pus are small and multiple, with a minimal amount of liquidity, and do not cause underlying brain necrosis per se. Purulent pachymeningitis may mean either of the two referred to above or may be another form of meningeal infection that is present as infected granulations in the subdural space with very little or no liquid pus. This is usually a transient, early form of subdural-space infection, which if untreated will go on to either subdural empyema or subdural abscess. The other names proposed are even less specific. The diagnosis of any of these conditions depends in the last analysis on the ability of the doctor to add untreated or inadequately treated infections of the ear, nose and throat, trauma to the head and such chronic infections as lung abscesses to the various manifestations of suddenly developing signs and symptoms of increasing intracranial pressure, localized irritation or destruction of the cerebral cortex, meningismus or what might be called mild meningitis, and to come out with the suspicion that some untreated infectious process is developing in the cranial cavity. Suspicion is confirmed and the diagnosis made by demonstrating pus or granulations in the subdural space at operation. The same signs and symptoms, except for the operative findings, may be caused by a brain abscess, an obstructive cerebral thrombophlebitis and the ordinary forms of meningitis. Experience, as mirrored in the literature at least, has shown that there is no such thing as a definite syndrome (such as that proposed by Kubik and Adams[238]) that will serve to warrant a differential diagnosis without operative investigation of the subdural space. The treatment calls for the constant provision of four fundamental procedures. The first is surgical drainage, which must, of necessity, be inadequate but which must be as adequate as possible and may have to be accompanied by decompression. The second, and this is carried out simultaneously with the surgical drainage, is intrathecal and intraventricular administration of penicillin together with the introduction of penicillin into the subdural space *after* the space has been cultured. Sulfadiazine is also started intravenously and by mouth. The third is the determination of the sensitivity of the infecting organism with a view of using the most lethal drug, whether penicillin, sulfadiazine, streptomycin, aureomycin, bacitracin or the like. The fourth is adequate surgical drainage of the original source of the infection. All are equally important, and no one can be safely omitted. To Cairns and his group at Oxford must go the credit for the development of the antibiotic therapy and to Le Beau of the late Clovis Vincent's clinic in Paris the credit for emphasizing and insisting on adequate therapy of the original source.[237,241-251]

Lobotomy

Lobotomy used in its generic sense to include all operations on the frontal lobes and the thalamus and designed as a means of surgically alleviating certain mental disease and otherwise intractable pain has been fully reviewed in the *Journal* by Greenblatt and Myerson[252] as recently as June, 1949. This therapy, so far as advances in neurosurgery go, ranks with the work done in the treatment of patients paralyzed because of injuries to

the spinal cord and cauda equina. Its application is more widespread but neither the most suitable among the methods advocated nor the final end results have as yet been crystallized to the same extent. The indications, however, have been well summarized by Tucker and Dynes[253] and are as follows: "1. There should be symptoms of agitation, depression or self-concern; 2. the condition (i.e. the mental disease) should be such that it could not be expected to respond to any other type of treatment; 3. the degree of suffering and incapacity must be sufficient to justify the operation; 4. the family situation should be favorable for rehabilitation of the patient; 5. the expected postoperative condition must be considered a sufficient improvement to justify the operation; and 6. there should not be organic brain disease." There seems to be general agreement that the clinical conditions that are apt to respond favorably to one or another of the various types of proposed operations are chronic agitated depressions, varieties of schizophremia with a large emotional or paranoid element, chronic severe obsessive compulsive neuroses, chronic severe hypochondriacal neuroses and the intractable pain of metastatic malignant lesions, particularly when associated with drug addiction. It is also worthy of note that the originator of lobotomy in man — Dr. Egas Moniz[254] — was recently awarded the Nobel Prize in Medicine.

Cranial Nerves

Fifth or Trigeminal Nerve

Surgery of this nerve is still chiefly concerned with the therapy of trigeminal neuralgia. In the last nine years the surgeons have been almost exclusively occupied with the best technical method of accomplishing this. The classic operation in which the sensory root, and frequently the motor root as well, was totally divided behind the ganglion has been modified to the extent that instead of a total section a differential section of the sensory root is done. The standard technic at present is to spare the sensory fibers leading to the first division as well as those of the motor root in patients whose pain is limited to the lower two divisions. The operation may or may not have been preceded by alcohol injection of the second and third branches in accordance with the predilections of the operator and the condition and wishes of his patient. Trichlorethylene, from which so much had been hoped at one time, has proved to be a total failure. A differential diagnosis between true trigeminal neuralgia and atypical facial pain — so-called — still presents great difficulty at times, but, in general, a diagnosis of true trigeminal neuralgia is still warranted if the pain occurs in attacks without warning and at unpredictable intervals and without cause, is invariably limited to the same side of the face and can be instituted by stimulation of a trigger point. The disease is remissive and patients may remain free of attacks for long intervals (one or two years) without any treatment, only to have the pain recur with its original severity and frequency. This has led some physicians to put unjustified faith in medical as opposed to surgical therapy. Experience has demonstrated that such therapy as the use of vitamins, although harmless, is ineffectual in control of the pain.

The classic operative approach to the sensory and motor roots has been extradurally via the temporal route. This is still the most popular. It requires division of the middle meningeal artery and entails danger of facial paralysis from compression of that nerve by hemorrhage in the fallopian canal. This is caused by operative rupture of the fallopian vein at the hiatus falopii on the anterior surface of the petrous bone. For these reasons this temporal approach has been modified by some.[255, 256] They operate intradurally, elevating the temporal lobe and locating the ganglion and the root in their dural sheaths by palpation of the floor of the skull. The only possible contraindications to this method seem to be the increased danger of contusion of the temporal lobe from direct pressure from retraction. Its ease depends on the amount of cerebrospinal fluid that can be emptied from the basilar subarachnoid space. If this is small, with resultant necessary excessive retraction of the temporal lobe, it may be better to proceed extradurally. With the intradural method the middle meningeal artery is not necessarily divided, and the fallopian vein not damaged. Differential section of the sensory root by a subtentorial approach was advocated by Dandy[257] as a substitute for the extradural temporal operation. Most surgeons find it a much more difficult technical procedure and use this approach only when some other cranial nerve has to be divided simultaneously. Finally, Sjörqvist[258] advocated division of the spinal tract of the fifth nerve in the medulla, claiming that such a division would control the pain but not alter sensation, especially of the cornea. Adequate experience[259] has demonstrated that the operation is inexact, does not live up to its claims, is frequently ineffective and may be technically extremely difficult. It is no longer considered a satisfactory method of treating trigeminal neuralgia. Complications still beset a small but significant number of patients operated upon by any route. The most feared and important are the corneal lesions. Pannabecker[260] reports on this from the late Dr. Peet's clinic at Ann Arbor. Corneal lesions occurred in 18 per cent of 878 cases treated from 1927 to 1943. Operations on the lower branches of the trigeminal nerve, which did not involve the first or ophthalmic branch, did not cause damage to the cornea. Corneal anesthesia and lagophthalmos gave the greatest incidence of trouble. These surgeons and all others who have to deal with such patients agree that if the fibers to the first branch are cut the eyelids of

the involved eye should be sutured at the time of the section. Dryness caused by lack of tears must also be guarded against. Proper care at once and for a sufficient time after the root section should eliminate such complications as extensive corneal scarring, loss of an eye and the like.

Postoperative paresthesias in the anesthetic area still occur, are unpredictable preoperatively, do not respond to treatment and usually either get well or diminish to a bearable level. Reoperation does not affect them.

Bilateral trigeminal neuralgia occurs in a small but significant number of patients. It usually develops first on one side of the face and then years later on the other. Since bilateral operation is perfectly feasible if one motor root is left intact its treatment depends primarily upon the care with which the first operation is performed.

The Facial Nerve

Facial paralysis is a not uncommon complication of neurosurgical procedures (such as removal of an acoustic neuroma) or of wounds of the face, after operations about the ear, as the result of exposure, or after extensive cerebral cortical damage from trauma[261] or excision of a tumor.

Central injury in every case and peripheral injury in many do not lend themselves to direct repair by methods leading to reactivation of the nerve. As a substitute many ingenious methods of "face lifting" have been proposed. Schuessler[262] used tantalum wire to connect the key paralyzed points to bundles of the still active temporal muscle. Hanrahan and Dandy[263] devised a modification of Brown and Gillie's earlier experience and described the use of fascial slings attached to the temporal fascia. Brown and McDowell[264] and Brown, McDowell and Fryer[265] employ fascial loops, which are so placed as to be acted upon by the temporal muscle.

Direct attack and end-to-end suture of the nerve at the point of division in the canal after the method of Ballance and Duel is advocated by Tickle[266] and Kettel[267] if not more than 1 or 2 cm. of gap exists. If more of the nerve is lost the canal is opened, and the nerve lifted out and rerouted prior to suture. This destroys the auditory mechanism but gains 2 cm. or more slack. Grafts from the anterior cutaneous branch of the femoral nerve have also been used. White[268] describes removal of the parotid gland for repair of an extracanalicular division, getting by this means 1.5 cm. of slack and making an end-to-end suture possible. Koechlin[269] would do an end-to-end suture in all fresh cases where feasible and if not feasible would interpose a graft. He has been disappointed in the results of anastomoses in long-standing cases and in that event prefers autoplasty with or without myoplasty.

Hypoglossal-facial anastomoses were covered in 1940 in a paper and discussion by Coleman[270] and a more recent paper by Trumble.[271] The results are no better than satisfactory, and there is always the question of associated movements to be considered if the reinnervation is successful.

Bell's palsy, or paralysis of the facial nerve without anatomic interruption, its pathology and surgical therapy are well covered in an article by Kettel.[272] He reports on 50 patients whom he has operated upon by the method of Ballance and Duel. He points out that from 85 to 90 per cent of these patients responded to conservative therapy, and believes that a decompression of the nerve should be performed whenever signs of returning function have not appeared in two months, if spontaneous recovery of mobility has ceased before complete restitution has been attained and if there is a relapsing paresis. He regards the pathology as that of an ischemia of the nerve and believes that the decompression allows for re-establishment of adequate circulation.

Ménière's and Related Syndromes of the Vestibular Apparatus

The discussion of the diagnosis and treatment of the various syndromes related to the vestibular apparatus in man has been much beclouded by lack of uniformity of definition of what particular syndrome the writer is discussing. The predominant symptom however is always a loss of the patient's sense of position in space complicated by vertigo, to which is added loss of equilibrium, nausea and possibly vomiting and, in certain cases, unilateral deafness and tinnitus. These are common to acute toxic or infectious labyrinthitis as well as to the more chronic Ménière's syndrome. Since the first is a short-term disease and the other a gradually progressive process the disease must be carefully defined whenever a certain therapy is advocated or studied for its remedial effect. This is especially true of patients whose disequilibration is that of neither the strictly acute labyrinthitis nor the classic, more prolonged Ménière's syndrome. Local disease of the middle ear of a mechanical type with, for example, a flapping drum or a closed eustachian tube as well as apoplectic attacks involving the same area are usually self-evident, and neither they nor tumors of the cerebellopontine angle (except in rare cases) should cause confusion. A good review of the problem of Ménière's syndrome was presented at the clinical Congress of Surgeons in 1940 by Dandy[273] and Tobey.[274] The effect of histamine,[275] alterations in the metabolism of water and salt, or potassium[276] have had considerable vogue as the basis for treatment of this condition. Selfridge,[277] in 1949, emphasized that the therapeutic aspect of this problem has reached the level of *reductio ad absurdum*. He points out that Ménière's disease has variously been ascribed to disturbances of the endocrine glands; infections; syphilis; low and high blood pressure; disturbances of the metabolism of

salt, potassium, magnesium, iodine and calcium; deficiencies of certain vitamins such as thiamine, nicotinic acid and choline compounds; allergy; creatine-creatanine metabolism; and the action of the nondecarboxylated fraction of histidine-histamine. Selfridge himself relates the disease to disturbances in nutrition. Medical therapy has been equally confused and has in general followed the lines indicated by this list of possible causes. The therapeutic possibilities of Dramamine have yet to be investigated. In addition to the accepted surgical treatment in which the entire eighth nerve,[273] or only the vestibular portion of the nerve; was divided,[278] Day[279] has accomplished the same total unilateral destruction of vestibular function by opening the horizontal canal through a mastoid incision. After this a needle is inserted into the canal until it enters the vestibule, which is then coagulated electrically. Lempert[280] describes a technic for effecting atraumatic and aseptic degeneration of the endolymphatic labyrinth by removal of the stapes and the round window of the labyrinth. Only 10 patients have been operated upon by this method.

It appears that in the past ten years no greater knowledge of the cause of chronic recurring disequilibration associated with advancing nonaural deafness and tinnitus has been acquired than in the previous decade. However, as noted above, the number of suggested causes has been multiplied, and treatment has varied accordingly. It seems that to cure the dizzy attacks of true Ménière's disease the vestibular nerve must be cut or the semicircular canals destroyed. All other therapeutic procedures either have not held up under prolonged testing or have not been in use long enough as yet. In particular it should be noted that the true Ménière syndrome should not be diagnosed and certainly should not be subjected to destructive therapy until the triad of nonaural deafness, tinnitus and explosive, unpredictable attacks of vertigo are present. Moreover, the last should have been present for at least eighteen months. Even then such procedures as repeated regular dilatation of the eustachian tube should have been given a fair trial first. The patient should also have received large doses of vitamins and an adequate diet and probably small regular doses of phenobarbital as well. Restriction of salt and the administration of sodium or potassium seems to be entirely useless. In particular it should be borne in mind that Ménière's syndrome is eventually a self-limited disease, that it has remissions varying from days to years, and that its etiology is at present unknown.

The Glossopharyngeal Nerve

An important paper by Ray and Stewart[281] describes 14 of their patients and 4 of Bracy's as having had a temporary rise in blood pressure and cardiac rate after intracranial division of this nerve for relief of pain. As the result of this and other experience, including 3 cases in which the ninth nerve was divided intracranially on one side for the relief of the carotid-sinus syndrome, this procedure is considered the ideal surgical means of correcting the symptoms of the variety of carotid-sinus syndrome that needs surgery.

Neurosurgery of Children

Exclusive of traumatic and tumor neurosurgical procedures, which are basically the same in children as they are in adults, neurosurgery in children concerns itself with the correction of congenital or immediately postnatal deformities of the central nervous system.

Not only because of its importance and frequency but also because of the authority inherent in the work, the series of papers on *spina bifida*, *cranium bifidum* and the *Arnold-Chiari syndrome* by Ingraham and his associates[282-286] are outstanding examples of progress in children's neurosurgery in the past nine years. No one attempting to deal with these difficult and discouraging deformities should fail to make himself thoroughly familiar with these articles. They describe not only what can be done but what can be expected after treatment. Other earlier articles on the Arnold-Chiari deformity are also helpful.

Endoscopic coagulation of the choroid plexuses[287] as a means of treating *congenital hydrocephalus* has not lived up to its earlier expectations. Many such patients have their hydrocephalus as an actual or potential accompaniment of a spina bifida or cranium bifidum, the Arnold-Chiari deformity or platybasia. The crux of the problem is the determination, prior to a final decision about treatability, of the amount of brain destruction and consequent amount of expected later development of cerebral function. The injection of dye into the cerebrospinal spaces and the visualization by air of the amount of cerebral cortex are essential preliminaries. Unfortunately, except when associated with the deformities mentioned above, any kind of therapy of the hydrocephalus itself is usually ineffective, and the outlook, regardless of treatment, discouraging in the extreme.

Ingraham and Campbell[288] have recorded 3 cases of the *Marcus-Gunn phenomenon*, a rare congenital affection in which congenital ptosis of one eyelid is accompanied by peculiar associated movements of the affected lid. In 1 of their cases the phenomenon was produced by cortical stimulation. They believe this to be the first time this has been done.

Platybasia. This congenital defect manifests itself in the second decade or after. It may also be acquired as the result of disease[289] and may be initiated by injury.[290] In the congenital and acquired forms it is generally agreed that active treatment is indicated whenever the neurologic symptoms are disabling or progressive. The symptoms are

produced by compression of the cerebellum, the brain stem, certain cranial nerves and the upper cervical cord either alone or in any combination. There is demonstrable diminution of the size of the posterior fossa, invagination of the occiput and upward displacement of the upper cervical vertebras. The diagnosis is made by x-ray examination. The congenital variety is frequently associated with other congenital anomalies. The treatment is surgical, a decompression of the compressed portion of the nervous system being provided.[289, 291] Syringomyelia and syringobulbia are not infrequently blamed for the production of symptoms actually traceable to the platybasia. It appears that, if looked for, the condition is less rare than has been supposed.

Premature synostosis of the cranium prevents normal growth of the contained brain, and if it is uncorrected, loss of vision and other serious sequelae may follow. Faber and Towne[292] report 5 cases of this condition in which provision for normal enlargement of the skull was made by the removal of strips of bone parallel to the closed suture or sutures. They call attention to the fact that relief must be afforded prior to the infant's attaining its sixth month, and this can best be done at the age of ten weeks. I believe this to be the first time that prophylactic surgery has been effectively carried out in this serious and not uncommon condition, thus marking definite progress. Ingraham, Matson and Alexander[293] report on the experimental creation of artificial sutures in animals and in 6 infants, polyethylene being the preferred substance for use in maintaining the separation of the cut edges of the bone. They follow this report with a paper on 50 patients,[294] 44 of whom were treated by surgical methods in line with their experimental work. The Faber and Towne type of craniectomy is performed, but instead of closing the wound without further procedures, the cut edges of the bone are covered with polyethylene film. They point out that the best results are obtained when surgical correction of the condition is carried out during the first year of life.

Pickles[295] reviews the literature concerned with *duplication of the spinal cord* (*diplomyelia*). He has collected 46 cases of this condition. To these he adds a case of his own, and I can also add 2 unpublished cases of mine.

Another congenital rarity that is worthy of mention is hereditary *stenosis of the aqueduct of Sylvius* as a cause of congenital hydrocephalus. Bickers and Adams[296] have collected 23 such cases from the literature and added a most carefully studied one of their own. This condition is not to be confused with acquired (postnatal) hydrocephalus from stenosis of the aqueduct. The patient of Bickers and Adams was one of 7 similar cases that occurred in two successive generations of one family.

Peripheral Nervous System

During the past nine years many attempts have been made to bring the surgery of injuries to the peripheral nervous system on to a more rational plane. The significant advances have been three, all therapeutic.

Technic of Repair

The first has been the reassertion that the earliest suture that will maintain undistorted, tensionless, approximately anatomic, close apposition of otherwise normal cut ends of nerves that have sustained a minimal amount of injury from the sutures used for this purpose will assure the maximum amount of functional return.[297] All neuromas, gliomas and scar tissue must have been excised prior to suture. Kirklin et al.[298] point out, as many others have done before them, that "complete recovery of function, to a degree comparable to the normal state, is indeed rare after nerve sutures." The optimum time for suture is within three months[298] and preferably within one month, but later repair up to fifteen months should not be denied the patient. Primary suture appears to be less advantageous from the point of view of maintenance and approximation of the suture line than early secondary suture if done within the first month.[279-299] If primary suture is decided against, however, it is essential that the cut nerve ends be anchored in place — preferably by radiopaque sutures — when the wound is first débrided or sutured. The suture material used seems — within limits — to make little difference, preference being given to silk and fine tantalum wire mounted in an atraumatic needle. It was first thought that tantalum would prove the most successful because of the minimal tissue reaction it set up locally,[300, 301] but for practical purpose silk, steel wire and cotton have proved as satisfactory as tantalum, the additional advantage of ease of handling tipping the scales in favor of the nonmetallic material. All neurosurgeons experienced in the field agree, however, that the sutured ends should be made radiologically visible by single tantalum-wire stitches, which can thus be used to determine the possible presence of a separated or pulled-apart suture line during supposed union without the necessity of re-exploration.[301, 302]

Another *sine qua non*, regardless of all other factors, is the need for adequate maintenance of the contractility and size of all paralyzed muscles, and the mobility of all immobilized joints by massage, passive motion and direct galvanic stimulation. Successful covering of the suture line in an endeavor to prevent the ingress of scar was thought to have been solved by the use of tantalum-foil wrapping.[299-301] The first world war had demonstrated the danger of using such materials as sections of blood vessels, Cargile membrane and fascia.

Unfortunately, the majority of the evidence is to the effect that although better than these latter and useful in a certain number of instances, tantalum-foil wrapping does more harm than good.[298] The best protection so far evolved is freedom from sepsis, minimal scar formation and burial of the suture line in the belly of a split muscle. Sutures should never transfix the cut ends of peripheral nerves even though so-called sling sutures have been advised. The justification for such a procedure lies in the need for approximation of the cut ends under tension, which of itself leads to failure of union even with the sling suture in place. In addition, such a suture encourages intraneural scar formation with its restriction of function at a site that would not otherwise be thus involved, and in an area proximal to the already existent point of damage.

Sutures should, if possible, be placed only through the epineurium. The fact that the epineurium thickens sufficiently during the first month after division and thus provides better holding ground for the suture material is the chief reason for preferring early secondary to primary suture. In this connection it appears that forcible stretching of the proximal segment of a divided peripheral nerve by traction sutures placed through the neuroma and attached with the limb in flexion to a fixed point below the joint that it crosses is of little use.[303] The amount of stretch possible in a peripheral nerve is not great and will be governed by the blood supply.[304, 305] This is easily damaged without the possibility of recognition by the surgeon. Necrosis of a previously viable peripheral segment of the nerve results. If approximation without tension cannot be accomplished by transposition or stripping of the branches of the divided nerve, or both, together with positioning of the limb, traction by way of the neuroma will not work either. Grafting may be tried under such conditions, but too much should not be expected from it. It appears that the only type of graft that has any chance of success is the heterograft. Grafts from other species or from other members of the same species will not succeed.[306-310] The smaller the nerve grafted, the better the chance of success. Cable grafts promise better success in the grafting of large nerves than grafting with segments of other large nerves.[299]

Experience with traumatic interruption of the deep or posterior interosseous branch of the radial nerve justifies the conclusion that repair can be done in most cases and that tendon transplantation is not justified as a primary procedure. Allbritten[311] reports 15 and Mayer and Mayfield[312] 58 cases. Aird[313] describes an operation that permits the exposure of high lesions of the sciatic, and Freeman[314] a combined approach to the obturator and femoral nerves. This is of use in dealing with certain minor spasms in paraplegic patients. The Livingstons and Davis,[315] Fahlund[316] and the Livingstons and Andrus[317] report on the technic of suture, on the suture of the posterior tibial nerve below the knee and on resection of the neck of the fibula in repair of the peroneal nerve.

Plasma Silk Suture

The second advance has been the successful union of the cut ends with the aid of clotted plasma and a minimum of suture material. The effective use of this method has been limited, but Bateman[318] records striking success in 350 cases. Singer,[319] Tarlov[320] and Tarlov and Benjamin[321] developed the method experimentally and clinically from work done by Young and Medawar[322] and Seddon and Medawar,[323] who worked with fortified cockerel plasma. The former three authors substituted autologous plasma and devised a method for depositing and holding the plasma in proper approximation in the suture line. Bateman added silk sutures. There can be no doubt that this method marks a great advance in the repair of divided peripheral nerves.

Injury to the Peripheral Sympathetic Nervous System

The third development has to do with the treatment of causalgia, Volkmann's contracture, traumatic arterial vasospasm and phantom limb. Smithwick[324] summarized the causalgia problem in this journal in 1949. It has been conclusively shown that effective preganglionic interruption of the sympathetic supply to the causalgic limb will cure the patient of his symptoms in over 90 per cent of the cases. This applies also to the minor causalgia of Homans,[325] as well as Sudek's atrophy[326] of the extremities. The diagnosis of causalgia as well as the appropriateness of the projected sympathectomy is made by the demonstration of temporary relief following sympathectomy by novocain injection of the appropriate ganglions. Bjorkenheim[327] has made clinical tests in various conditions, and Berry et al.[328] have advocated and reported success with tetraethylammonium used in place of injections. Shumacker, Speigel and Upjohn[329] have published particularly good articles on this subject.

Smithwick[330] reviewed the status of peripheral vasospasm in 1941. Foisie[331] and Console,[332] however, have extended this to include Volkmann's contracture and traumatic arterial vasospasm and have shown the intimate association between these conditions and the sympathetic nervous system. Here, too, the diagnosis and treatment have proved to be traceable to abnormal activity of the peripheral portion of the sympathetic system. Sympathectomy, whether by chemical block of the ganglions, preganglionic interruption or peripheral interruption as accomplished by resection of the spastic part of the involved artery, is specific. In particular, Volkmann's contracture, the symptoms of which were hitherto believed to be traceable to compression of the brachial artery by clot

under the bicipital and antecubital fascia,[333] has been shown by Foisie[334] to be actually the end result of "nearly complete ischemia produced by arterial spasm of the main artery and the collateral circulation." Interruption of the sympathetic reflex arc or in stubborn cases excision of the spastic segment of the involved artery, which accomplishes the same thing peripherally rather than centrally, will cure the condition in all cases if it is performed sufficiently early. Cahuzac and Jung[335] and Parkes[336] have presented additional evidence in support of this point of view. In view of the constant history of injury, the constant implication of abnormal activity of the sympathetic nervous system with resistant vasospasm and the constant relief of symptoms by interruption of the sympathetic arc, whether temporary or complete and whether central or peripheral, it appears to be worth while and in the interest of more efficient therapy to class those conditions now held to be separate entities under the names of major causalgia, Homans's minor causalgia, Sudek's atrophy, Volkmann's contracture, and segmental arterial spasm under the all-inclusive heading of injury to the peripheral portion of the sympathetic nervous system.

Phantom Limbs

The therapy of phantom limbs has not as yet reached a point where it can be considered satisfactory. However, certain factors appear to be fairly constant. There are at least three elements involved. They include the signs and symptoms caused by any amputation neuromas that may be present. These are fairly characteristic and are associated with irritation of the bulbous nerve end. If uncomplicated they can be relieved by resection, crush and ligation [337] of the distal cut end of the normal portion of the nerve. There are also the symptoms and signs of abnormal functioning of the sympathetic nervous system. These are analogous to those described above under injury to the peripheral part of the sympathetic nervous system. They are treated as described above if they are uncomplicated, which they usually are not. Finally, there are the painful spasm and forcible contractures of portions of the amputated limb. If the condition is uncomplicated it appears at present that this aspect of the problem is best treated by the dorsal cordotomy of Browder and Gallagher[338] and Pool.[339] Unfortunately, the usual patient with a phantom limb presents a mixture of all three of these entities, and this already confused picture is still further confused in the majority of cases by a neuropsychiatric overlay. Other therapeutic methods have included high cervical anterolateral cordotomy,[340] resection of the sensory cortex,[341] prefrontal lobotomy,[342] electroconvulsive therapy[343] and abolition of stump pain by local percussion (without bruising) of the stump with a piece of wood, in addition to the application of pressure locally after the production of hypalgesia by injection of procaine locally or the application of a tight sphygmomanometer cuff.[344] None of these methods have been used with sufficient frequency to be taken out of the experimental class as yet.

Neuromas

In my experience the present preferred method of preventing the formation of neuromas on the distal cut ends of the proximal segment of divided peripheral nerves is still that devised by MacMurray while working in Sir Robert Jones's clinic in Liverpool during the first world war, and more recently advocated by Herrmann and Gibbs[337] and Cieslak and Stout.[345] The neuroma is resected, the cut end of the nerve crushed, and the nerve ligated at the crushed area with unabsorbable suture material. Alcohol may or may not be injected into the nerve proximal to ligature. Insertion of the cut end of the nerve in adjacent bone[346] and White's[301] proposal that the end be enclosed in a crushed and folded wrapping of tantalum foil may also be tried. Teneff,[347] of the University of Turin, Italy, reports success in the prevention of amputation neuromas by implanting the cut end of the proximal segment into nearby muscles. He discusses 6 successful cases. Experimentally, he found in rabbits that about twenty-five days after implantation neurofibrils penetrate the narrow zone of loose scar tissue in all directions, separate into fasciculi and filter into the muscular and between the adjoining connective and intrafibrillar tissues. In sixty-five to seventy days single neurofibrils have separated and become firmly attached to muscle fibers without a trace of connective-tissue proliferation. Regardless of any other factors, if the cut end is untreated a neuroma with all its attendant later symptoms will form no matter what the size of the nerve may be.

Brachial Plexus

A good review of the various aspects of brachial-plexus surgery has been written by Davis, Martin and Perret.[348] They emphasize again the need for early surgery. This should be done as soon as the deleterious effects of any associated injury to the soft parts have disappeared. In the interval too great importance cannot be attached to adequate physiotherapy and splinting of the arm and hand in antigravity positions. Many nerve fibers that appear at first to have been torn will prove after proper physiotherapeutic care to have been merely stretched and to be capable of resumption of function without further treatment. In my experience judicious radiotherapy over the supraclavicular area has also proved helpful, particularly when a considerable hematoma has formed about the plexus. It appears to have a definite effect in lessening the formation of compressive scarring. This general type of conservative treatment is of par-

ticular use in the brachial-plexus injuries of the newborn. In all such cases the damaged arm should be promptly and constantly maintained in shoulder abduction, forearm pronation and, if possible, extension of the wrist and fingers. The elbow should be slightly flexed. Fischer[349] has devised a bonnet with a cuff attached in such a way as to anchor the wrist and thus maintain this optimum position. The wrist can also be fastened to the head of the crib, but this involves fixation of the baby's body as well. Nothing can be done surgically for torn roots, and very little for tears of the primary cords. However, the adult patient, in particular, should not be denied the possible advantages of more accurate estimation of the extent and place of the damage. This is only possible by an exploration of the plexus. The chance of repair is enhanced in direct relation to the amount and efficiency of the preoperative physiotherapy. Operation should not be delayed for longer than four to six weeks after injury unless signs of resumption of function manifest themselves and steadily increase. Neurolysis and end-to-end suture (after neuroma and glioma resection) between normal nerve tissue is the procedure of choice. The clavicle may be divided, turned out of the way and repaired by wiring if necessary to get an adequate exposure. Indeed, Elkins has shown in his surgery of wounds of the great vessels in the base of the neck that the clavicle may be completely removed and not replaced without any obvious functional or cosmetic deformity in men. There seems to be good reason to suppose that there will be less cosmetic deformity after such a procedure than after most wirings. For plexus tears that cannot be repaired as above because of scarring or wide separation of the nerve ends, Lurje,[350] of Moscow, has suggested the ingenious method of implanting the functionally viable cut end of the proximal part of a previously undamaged motor nerve that is innervating a relatively useless muscle into the freshened proximal cut end of a hitherto irreparably damaged nerve that will, if regeneration can be brought about, again innervate and restore motion to an important muscle. For example, he reports having used the undamaged long thoracic and anterior thoracic as well as one of the tricipital branches of the radial nerve to reactivate the suprascapular, the musculocutaneous and the axillary nerve. Thus, by partially sarcificing the functions of the pectoralis and part of the triceps, as well as all the serratus anterior muscles, he reports having restored function to the previously paralyzed deltoid, biceps and supraspinatus muscles. Such an operation requires the most painstaking type of preoperative estimation, accurate stimulation at the time of operation and muscles whose contractile qualities have not been irreparably damaged by neglect, scarring and fibrosis. The procedure is unquestionably worthy of more investigation.

Diagnosis

Livingston[351] has reduced the problem of identification of injured peripheral nerves to its simplest terms. This is important because it provides a reliable short method that should go far toward eliminating the dangers of missing the presence of a peripheral nerve injury when it is part of an extensive laceration of an extremity. In the upper extremity the radial, median and ulnar nerves are tested as follows: the radial, by the ability to extend actively the distal phalanx of the thumb, the median, by the perception of pinprick over the palmar surface of the distal phalanx of the index finger, and the ulnar by the perception of pinprick over the surface of the fifth finger. If there is an injury to the palm itself all fingers must be separately tested for perception of pinprick. Lesions of the brachial plexus can be identified as variable combinations of these three major nerves. In the leg the function of the tibial and peroneal nerves is tested as follows: the peroneal, by the ability to dorsiflex the great toe provided the motion is started from a neutral position, and the tibial, by the ability to plantar-flex the great toe, again starting the test with the toe in the neutral position. In wounds below the knee the tibial function must be further checked by testing for sensation in the anterior plantar area. Since anesthesia in the anterior plantar area is at least as disabling as and in most cases actually more disabling than paralysis of the muscles supplied by the tibial nerve, this latter test is of great importance.

Pain

The specific types of pain such as trigeminal neuralgia, glossopharyngeal neuralgia and the pain of an amputation stump and causalgia have been dealt with in previous sections. There are, however, methods besides lobotomy and topectomy that are useful for treating what might be called nonspecific pain.

Pain in cancer of the face, jaws and neck has come in for considerable study in the last nine years. Munro[352] suggests that early denervation of the diseased area should be done and that postponing such denervation leads not only to unnecessary pain but also to inefficient surgery. He reports 30 cases in which only 2 patients were alive and had relief at the end of any significant follow-up period. Another approach to this problem as well as the problem of other nonspecific pain in the upper extremities was made by Schwartz and O'Leary.[353] They advocated section of the spinothalamic tract at the level of the inferior olivary body. This was after Grant and Weinberger[354] had reported on a follow-up study of 15 cases treated for trigeminal neuralgia and cancer by medullary tractotomy. Tractotomy of the medulla for pain in the shoulders and above was not so successful as had been hoped,

however. Adams and Munro[355] demonstrated by post-mortem studies on 3 patients who had had medullary tractotomies performed on them that "it was impossible to cut the cervical portion of (the spino-thalamic) tract in the medulla, without causing crippling and unjustifiable damage." In the same year Walker[356] described his operation for mesencephalic tractotomy. He claimed that pain and temperature fibers were divided but that the tactile and proprioceptive fibers were left intact. The operation was performed on 2 patients. One died as the result of the operation, and the other obtained relief of her pain. In a third patient the operation had to be abandoned before the section was made.

The paraplegic patients present their own peculiar pain problems, which are only now beginning to be understood. They are of the root and vasospastic types, painful spasms and unidentifiable pain. Munro[22] has shown that among veterans the root type of pain is frequently relieved by a decompressive laminectomy, and if that does not work a high cordotomy will be effective provided the roots involved are those of the cauda equina. Vasospastic pain responds to appropriate sympathectomies, and painful spasm to anterior dorsolumbar rhizotomy if it is in the legs. Non-neurosurgical methods should not be neglected either. The removal of a suprapubic tube from the bladder or a piece of shrapnel from where it lies against a peripheral nerve or the spinal cord, the drainage of a deep abscess and the correction of intra-abdominal disease have all proved their effectiveness in the relief of what appeared to be neurogenic pain.

Although it is certainly true that one cannot, as yet, expect to relieve all types of pain in every case by neurosurgical methods, at least more weapons are at hand, as well as a better understanding of how to use them, than were available nine years ago. By and large the degree of success attained in relief of pain is directly proportional to the accuracy of its classification preoperatively and the accuracy of the choice of surgical procedure. It appears that the pain of radiculitis whether of the upper or lower extremity, if it cannot be relieved otherwise, is best treated by an anterolateral cordotomy at the second dorsal segment if the involved root or roots are below the ninth dorsal segment. Otherwise a high cervical anterolateral cordotomy with section of the upper posterior cervical roots should be performed. Relief of pain by the division of other posterior roots is a useless procedure and should not be done. The prognosis in regard to successful therapy of unclassifiable pain is, to say the least, questionable.

Surgical Therapy of Tremors, Rigidity and Festination

In 1941 Russell Meyers[357] covered the knowledge available at that time concerning the need for surgical therapy of tremors, rigidity and festination. He pointed out the impossibility of using animals for this branch of surgery and the fact that at that time the problem had already been attacked on four levels — that is, the spinal cord, the cerebral cortex, the basal ganglions and the cerebellomesencephalic complex. He postulated an ideal operation and discussed the pathogenesis of Parkinsonism. Eight cases that had been operated upon at the basal-ganglion level were reported. In the following year he added 2 more cases[358] and discussed the technical considerations connected with his operation, which was designed to interrupt the pallidofugal fibers. More recently and despite the disappointing earlier results from the same procedures, Oliver[359] has advocated lateral tractotomy with a division of the pyramidal tract at the level of the second to third cervical segments, and Ebin[360] a "combined lateral and ventral pyramidotomy" in the cervical cord. This is a very extensive procedure that crosses the midline and divides transversely not only the long and short tracts in the lateral white matter but also the majority of the gray matter of the cord, only small segments of the anterior and posterior portions being excluded. Eleven cases are reported. This operation must still be regarded as in the experimental stage.

Miscellaneous Considerations

Spastic children have been the subject of therapy with curare[361] and Tolserol.[362] The reports included only 6 patients treated with curare and are inadequate as a basis on which to draw conclusions. This is especially so in view of the overenthusiastic early reports of the curative effect of this drug in other types of spasticity and the demonstration[363] that any continued effect was psychologic and not pharmacologic. Tolserol was reported as having been used in 16 children with cerebral palsy under adequately controlled observation. The authors report that "no definite over-all improvement was noted."

Beck, McKhann and Belnap[364] have reported on 18 children in 11 of whom symptoms of *inadequate vascularization of the brain*, such as convulsions, spasticity and mental retardation, have been improved after a shunt operation devised by them. In this procedure an anastomosis is made between the common carotid artery and internal jugular vein on the right. Although not strictly a neurosurgical procedure it opens up neurosurgical possibilities that warrant its inclusion in this review.

Neurologic lesions that have neurosurgical implications because of the possibility that active surgery will be entertained as needed therapy in some of the possible differential diagnoses have been described by Adams, Cammermeyer and Denny-Brown,[365] Denny–Brown and Sciarra[366] and Hyslop.[367] The first by Adams et al. concerns 4 cases of acute *necrotizing hemorrhagic encepha-*

lopathy. Signs of intracranial infection following antecedent respiratory or gastrointestinal symptoms and evolving into headaches, hemiplegia, quadriplegia and stiff neck with an abnormal cerebrospinal fluid raised the question of metastatic intracranial infection of a surgical type. Death occurred in one or two days after the acute stage developed, and no surgical lesion was found at autopsy.

Colicky pains in the abdomen with nausea, vomiting and burgundy-red urine associated with signs that mimicked acute toxic delirium or ascending paralysis of the Landry type and were accompanied by convulsions and bulbar neuritis led to a diagnosis of *porphyria* in 2 cases extensively studied by Denny-Brown and Sciarra.[366] They stress the importance of entertaining this diagnosis in states of acute delirium.

Hyslop[367] has presented a detailed description of the effect of *electrical injuries on the nervous system*. Much useful information is contained in the paper.

It is notable that the last nine years have witnessed the definite emancipation of neurosurgery from the strictly limited field of tumor surgery. The development and application of the technics evolved by such pioneers as Horsley, Cushing, Frazier and Elsberg to the field of traumatic neurosurgery, as well as the more recent extension of the specialty into abnormal neurophysiology, now overshadow and dwarf the original efforts to deal with neoplasms of the nervous system. As this advance continues it becomes more and more apparent that to be a successful specialist in the restricted field of neurosurgery one has to have and apply an ever-increasing knowledge of the broad field of general surgery.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

BENJAMIN CASTLEMAN, M.D., *Associate Editor*

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CASE 36181

PRESENTATION OF CASE

A fifty-five-year-old landscape gardener entered the hospital because of abdominal pain.

About noon of the previous day, approximately thirty hours before admission, the patient experienced aches and pains over the entire body and felt extremely fatigued. He went to bed and slept intermittently until 2 a.m., sixteen hours before admission, when he awoke suddenly with a steady epigastric ache, somewhat greater on the right. He felt nauseated and vomited several times, and was seen by a physician at 7 a.m., when 16 mg. of morphine was given. The pain continued during the morning, the patient vomited occasionally, and he was seen again by his physician; the dose of morphine was repeated, and hospitalization advised. Shortly before admission he had a small bowel movement.

There was no history of previous illness. The patient's wife had had a prefrontal lobotomy, and the patient was considered to be "more than an average drinker" by his physician.

On physical examination the patient was rather drowsy and responded poorly. The pupils were constricted but reacted to light. The heart was slightly enlarged to the left, and a Grade I diastolic pulmonic murmur was heard. The lungs were clear and resonant. The abdomen was protuberant, moved with respirations and showed marked spasm in the upper portion, more so on the right. Slight minimal spasm was present in the lower abdomen. There was no cough or rebound tenderness; peristalsis was not heard. Rectal examination was negative.

The temperature was 99°F. and the pulse and respirations were normal; the blood pressure was 130 systolic, 70 diastolic.

Examination of the blood showed a hemoglobin of 15.1 gm. and a white-cell count of 17,200. Urinalysis disclosed a specific gravity of 1.010, a + test for albumin and a light-green sugar reaction. A stool specimen was guaiac negative. The serum amylase was 130 units. An x-ray film of the abdomen taken in the upright position revealed no free gas in the peritoneal cavity.

Following admission to the hospital dextrose was given intravenously, and a conservative course decided upon, with antibiotics and gastric suction. During the first twenty-four hours, in spite of about 3500 cc. of fluid injected intravenously, the patient excreted only about 100 cc. of urine. Re-examination about twenty-four hours after entry revealed discoloration and venous distention of the upper abdomen and resonance to percussion over the liver. Another x-ray film taken in the upright position confirmed the presence of a bubble of gas beneath the diaphragm. The fasting blood sugar at the time of death was 600 mg. per 100 cc. The patient showed no signs of improvement, the temperature stayed near 100°F., and he died about thirty-six hours after admission.

Differential Diagnosis

Dr. Henry H. Faxon*: Could we see the x-ray films?

Dr. James J. McCort: On the chest film the right leaf of the diaphragm is elevated. There are plate-like areas of atelectasis in the base of the right lung and a small amount of fluid in the right costophrenic sinus. In the plain films of the abdomen taken in the upright and supine positions, it is noted that there is gas in the large bowel extending from the cecum to the midportion of the descending colon. The large bowel is not dilated. There is no small-bowel distention, and no gas is seen in the stomach. On these films, there is no evidence of free air beneath the diaphragm. A film taken twenty-four hours later is said to have shown air beneath both leaves of the diaphragm, but this film is not available.

Dr. Faxon: It was stated that at the first x-ray examination there was no gas beneath the diaphragm. Was there a subsequent film?

Dr. George L. Nardi: A film taken twenty-four hours later showed a definite layer of gas; I saw it myself.

Dr. Faxon: Is there any gas in the transverse colon?

Dr. McCort: Gas is present in the ascending and transverse colon.

Dr. Faxon: I am going to start with the assumption that this was a rather typical case of acute pancreatitis and in the discussion will try to bring out the reasons why I believe this to be true and will mention the chief differential diagnoses, as well as try to reconcile some of the inconsistent facts in the history with the diagnosis of pancreatitis.

The serum amylase, although a single determination, was 130 units, which is definitely elevated by this hospital's standards. This determination was done approximately twenty-four to thirty hours after the onset of the disease, which is the period in which the amylase can most consistently be expected to be elevated. If it were taken too early in the course of the disease it might not have shown any elevation, and on the other hand if the determination were delayed forty-eight to seventy-two hours it might again have returned to normal. One cannot say that an elevated serum amylase is pathognomonic of acute pancreatitis, but certainly it is the most significant single finding one can have. Often we are too ready to disregard this test because we do not think the clinical picture fits the diagnosis of pancreatitis, but in this case both the amylase test and the clinical picture support the diagnosis of acute pancreatitis as the main lesion.

Acute pancreatitis is not a single pathologic entity but is divided into three main subdivisions: interstitial, suppurative and hemorrhagic. Interstitial pancreatitis comprises three fourths of the cases, with a mortality of about 5 per cent. Suppurative pancreatitis, which often goes on to the formation of a mass, comprises about 12 per cent of the cases and has a mortality that runs around 25 per cent. Acute hemorrhagic pancreatitis comprises the rest of the cases and is a decidedly lethal disease; regardless of the type of treatment, the mortality runs about 80 per cent in most of the reported series. It is the acute hemorrhagic pancreatitis that I think this man probably had. He was in the proper age group, the average age being about fifty.

The pain that this patient had was steady, severe and primarily upper abdominal and was associated with vomiting, which, as in most cases, was reflex rather than due to obstruction. Steady upper abdominal pain associated with almost immediate reflex vomiting is strongly suggestive of acute pancreatitis. The spasm was chiefly in the upper abdomen in the region of the lesion, as one would expect, and the tenderness and spasm in the lower part of the abdomen were undoubtedly due to the gravitation to that region of irritating peritoneal fluid.

It is stated in the record that the patient was "more than an average drinker," and it has frequently been brought out that alcohol is a predisposing factor in the onset of this condition.[1, 2] The supposedly typical attack of acute pancreatitis is in a person of approximately fifty who has had a heavy meal and a number of highballs. In this case it is rather interesting that the first attack apparently occurred at noon. Whether it was before or after his meal I do not know.

On entry the temperature was 99°F., and at the time of death it was approximately 100°F. The white-cell count was 17,200. The combination of only a slightly elevated temperature and a high white-cell count — and often it goes much higher than 17,000 — is typical of acute hemorrhagic pancreatitis.

*Chief surgeon, Cushing Veterans Administration Hospital; associate visiting surgeon, Massachusetts General Hospital.

Peristalsis was not heard, suggesting a paralytic type of ileus seen in a third to a half of the cases of hemorrhagic pancreatitis. The plain x-ray film is not striking but bears out this condition in part. It has been brought out in many reports[1,2] that paralytic ileus, as evinced by x-ray films, is likely to be segmental and that often the transverse colon shows more dilatation than other parts of the gastrointestinal tract. The absence of peristalsis regardless of the fact that the x-ray films show only slight dilatation suggests a paralytic ileus.

The urine gave a light-green test for sugar — sugar is often present in these cases and does not mean of necessity that diabetes was present beforehand. At the time of death the blood sugar was 600 mg. per 100 cc. — such high levels can occur in acute pancreatitis. If a patient recovers from the interstitial or suppurative pancreatitis, he may have a subsequent diabetes, but it is generally accepted that diabetes is not a predisposing factor in acute pancreatitis.

The discoloration of the upper abdomen is mentioned but is not too well described here. Cullen's sign, discoloration around the umbilicus, is seen in a number of conditions due to hemorrhage in the peritoneal cavity. At times in pancreatitis a "peculiar cyanosis" is often mentioned and is ordinarily attributed to a state of shock that these patients sometimes suffer. In this case I suspect that it was a Cullen's sign — if I am defining that sign correctly — and a manifestation of the hemorrhagic pancreatic disease. I am a little disturbed by the venous distention.

Dr. Nardi: This was not a Cullen's sign, but rather an atypical cyanosis.

Dr. Faxon: It is helpful to have this clarified and takes out of my mind the possibility that this whole picture was on the basis of an extensive venous thrombosis giving rise to a paralytic ileus and even possibly shutting off the renal blood supply. From what Dr. Nardi says I can discard this possibility.

One further note mentioned in the history is that the patient had a small bowel movement after the onset of symptoms. Sometimes in these cases there is actually a diarrhea, which is supposed to be caused by irritation of the autonomic system; whether or not it is too fanciful an explanation I am not ready to say.

What may have been considered by way of differential diagnosis when the patient was admitted? The three main things that are usually confused with this condition are perforated ulcer, gall-bladder disease and mechanical intestinal obstruction. So far as a perforated ulcer is concerned, I think that probably the reason the original x-ray film was taken was to look for gas under the diaphragm. If gas was seen there at the start, it would certainly have strongly suggested a diagnosis of perforated ulcer. It seems to me that if the physicians were equipped with the amylase determination at that time they would have had to assume that this man had pancreatitis but were seeking out some other condition in addition, such as a perforated ulcer, to explain the pneumoperitoneum. The onset was not altogether typical of either perforated ulcer or pancreatitis in that it started more slowly than either of those diseases do as a rule. If the severe pain that awakened the patient in the middle of the night had been his first symptom, it would have been more typical of both conditions. Mechanical intestinal obstruction would not occur without hyperactive peristalsis and without more signs of a dilated bowel. Unless the mechanical obstruction were very early, the pain would have been colicky, and localized in the upper abdomen if it were of the small bowel. I think we can rule out such obstruction.

Against gall-bladder disease is the fact that there was no biliary colic, and no reference of radiation of pain. Approximately a third to a half of the patients with acute pancreatitis have gall-bladder disease. It is surprising to me that in the group of cases reported by Paxton and Payne[1] 10 per cent had stones in the common duct. I see little to justify a diagnosis of gall-bladder disease here, although I think on a statistical basis there is a 50 per cent chance there was some biliary disease.

The gas beneath the diaphragm on x-ray examination twenty-four hours after entry is not too inconsistent with our diagnosis of pancreatitis, for a certain definite group of these patients will have ulcerations of the gastrointestinal tract as part of their disease. I think not only that the patient had acute hemorrhagic pancreatitis but also that it contributed to a perforation of the gastrointestinal tract, probably the duodenum as the most likely site statistically. Assuming that there was gas beneath the diaphragm, the only way it could have arrived there was either by an infection with a gas-producing organism or by a rupture of a viscus that contained gas. I see little to make me suppose that a gas-forming organism was the cause of the pancreatitis. In one series[2] in the literature 6 per cent of cases with pancreatitis showed peptic ulceration, and in another series[1] approximately 10 per cent of the patients had some gastrointestinal-tract bleeding.

There were certain other things in the history apparently unrelated to the pancreatitis that I will mention briefly. One is the relatively scant output of urine. I do not see why with 3500 cc. of fluid given intravenously the patient did not excrete more than 100 cc. of urine. Unless the gastric loss was tremendous he must have had renal damage to explain this finding. He should have excreted at least 500 cc. if there was an adequate fluid intake so that I think I will have to assume that he had some kidney damage as an incidental condition.

I think the fact that he died so promptly was consistent with the usual course of acute hemorrhagic pancreatitis, and the fact that he was treated con-

servatively outside and in the hospital leads me to suspect that this was the diagnosis of those treating him. I think he had, in addition to acute hemorrhagic pancreatitis, perforation of the gastrointestinal tract, a certain amount of kidney damage and about a 50 per cent chance of having gall-bladder disease.

DR. NARDI: The reason the x-ray film was taken the second time was that there was resonant percussion over the liver. The film showing air under the diaphragm was seen by the medical service as well as by our service, but I cannot remember whether a roentgenologist was present or not. Dr. Buttles, who did the autopsy, said he heard gas escape when he opened the abdomen.

DR. BENSON ROE: I did not hear the first part of the discussion, but I think it is only fair to mention that it was the impression of the service that he did have a perforated viscus and little clinical evidence, at first, to indicate an acute pancreatitis. He was treated conservatively, largely because of the time factor, and later we were glad we had not operated.

CLINICAL DIAGNOSES

Perforated peptic ulcer.
Peritonitis.

DR. FAXON'S DIAGNOSES

Acute hemorrhagic pancreatitis.
Perforated peptic ulcer.
Kidney damage.

ANATOMICAL DIAGNOSES

Acute hemorrhagic pancreatitis, with pneumoperitoneum.
Peritonitis, acute, with fat necrosis.
Pylonephritis, chronic.
Benign prostatic hyperplasia.
Hydroureter and hydronephrosis, bilateral.

PATHOLOGICAL DISCUSSION

DR. BENJAMIN CASTLEMAN: As Dr. Nardi mentioned, there was a large gush of air when the abdominal cavity was opened, and on further exploration we found about 400 cc. of cloudy fluid in the abdominal cavity. When the omentum was raised there were small bubbles of gas right over the pancreas. Careful examination was made of the stomach, duodenum and intestinal tract, but no evidence of a perforation was found. On examining the pancreas further, we found that about 95 per cent of it was destroyed by a very hemorrhagic necrotic process quite characteristic of the hemorrhagic type of acute pancreatitis. Culture of the pancreas yielded only colon bacilli and hemolytic streptococci, although a smear did show some gram-positive rods. We know that the colon bacillus does produce gas, and that may have been the answer here. It is interesting that the common bile duct and pancreatic duct opened together in the ampulla so that there was ample opportunity for a reflux of bile into the pancreatic duct to produce a pancreatitis. There was no gall-bladder disease. The kidneys were small and somewhat sclerotic, and the pelves were distended, as were both ureters — one measuring almost 2 cm. in circumference. On microscopical examination the kidneys showed extensive chronic pyelonephritis, probably caused by an enlarged prostate, which the patient had. We did not find anything that could be called a lower-nephron nephrosis.

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2. Siler, V. E., and Wulsin, J. H. Acute pancreatitis. *J. A. M. A.* **142**:78-85, 1950.

CASE 36182

PRESENTATION OF CASE

A seventy-eight-year-old nulliparous widow entered the hospital because of abdominal pain.

During the two years prior to admission the patient had experienced bouts of intermittent, crampy abdominal pain centering in the left lower quadrant and accompanied by nausea without vomiting. The episodes usually lasted a day or so. On occasions the pain traveled down the anterior aspect of the left thigh but never to the back or shoulder. In the six months before admission the attacks had recurred somewhat more frequently — every one or two months. The last one prior to the present had occurred five weeks before admission and was similar but less severe. Three days prior to admission after several days of vague abdominal discomfort, the patient developed pain in the left flank along with the usual left-lower-quadrant pain, which became increasingly intense. A maximum temperature of 102 to 103°F. was noted, without chills. Although she had been chronically constipated for years diarrhea or bloody or dark stools were denied.

The patient was not known to have had any other severe illnesses in the past. Many years previously she had experienced occasional attacks of right-upper-quadrant pain radiating to the interscapular region, without jaundice. She reported ankle edema at intervals during the past two years, without dyspnea. There had been a cough productive of small amounts of mucoid sputum for many years. She stated that she had observed very small amounts of thin, brownish vaginal discharge for "years." This discharge had not changed recently. The menopause was recalled at about the age of forty-eight years.

Physical examination revealed a pale although well developed and well nourished woman. The abdomen showed a generalized tenderness that was a little more marked in the left lower quadrant without spasm or masses. Peristalsis was quiet. Pelvic examination revealed a small cervix with a polyp presenting at the os. A mass indefinitely related to the uterus, which seemed to be anterior, could be felt just above the peritoneal floor on the left side. The mass could be made out only vaguely but proved tender on both vaginal and rectal examinations. A small amount of thin, brownish vaginal discharge was present.

The pulse was 80, and the respirations 20. The blood pressure was 165 systolic, 90 diastolic.

Urinalysis showed a specific gravity of 1.010, a + test for albumin, and a negative sugar reaction, and the sediment contained 12 white cells and a rare red blood cell per high-power field. Examination of the blood disclosed a white-cell count of 15,200, with 83 per cent neutrophils. The hemoglobin was 12.2 gm. Two stool guaiac tests were negative. The nonprotein nitrogen was 20 mg. per 100 cc. A chest roentgenogram revealed emphysema and cardiac enlargement both to the right and to the left. An abdominal film demonstrated an irregular calcification (1.7 by 1.0 cm.) in the bony pelvis slightly above the level of the ischial spine in the midline. There was a vague suggestion of a mass within the left portion of the bony pelvis, and it was thought that the uterus might be enlarged slightly. There was normal distribution of gas within the large and small bowel. A barium enema visualized the bowel up to the splenic flexure. A fair number of diverticula were noted in the sigmoid and descending colon. The lumen of the bowel was not narrowed. A vaginal smear was reported negative. An electrocardiogram was normal.

After admission the abdominal pain persisted along with the tenderness, mass and induration within the left pelvis. The patient became more comfortable, however, several days after the institution of penicillin and streptomycin therapy. Nevertheless, the temperature continued to spike to 101°F. daily.

On the eleventh hospital day an operation was performed.

Differential Diagnosis

Dr. Joe V. Meigs*: "A mass indefinitely related to the uterus, which seemed to be anterior" — may I ask exactly what this sentence means? Was the uterus anterior or was the mass anterior?

Dr. John B. McKittrick: The uterus was anterior in its relation to the mass.

Dr. Meigs: Are the x-ray films of any consequence?

Dr. James J. McCort: On the plain film of the abdomen an ill defined soft-tissue mass is seen in the left lower quadrant. There is also the suggestion of another rounded soft-tissue density adjacent to this mass, which may be the body of the uterus. On the barium-enema examination a large number of diverticula were found in the sigmoid and descending portion of the colon. A few diverticula are apparent in the ascending and transverse colon. In the sigmoid the mucosal folds are wide. At one point on the inferior margin of the sigmoid there is a small collection of barium, which may be outside one of the diverticula. This is shown best on the spot film. The examination of the chest shows only changes consistent with the age of the patient — bilateral emphysema and sclerosis of the aorta.

Dr. Meigs: Is it fair to ask if it could be diverticulitis by x-ray examination?

Dr. McCort: I think the changes are suggestive. The wide, coarse mucosal folds and the small collection of barium on the inferior margin of the sigmoid are consistent.

Dr. Meigs: In a woman seventy-eight years of age who has definite diverticulosis one would quite definitely think in the present circumstances of a diverticulitis. The temperature of 101 or 102°F. and the white-cell count are consistent with it, as is the tender mass, and the past history is

*Chief of gynecology, Massachusetts General Hospital.

consistent with attacks of diverticulitis as evidenced by two years of difficulty in the left lower quadrant. One can also consider that a woman this age may have had some sort of lesion in the adnexa on the left side. It is possible that a tumor of the ovary twisted and untwisted. Had this lesion been anterior to rather than behind the uterus, a dermoid cyst that was twisting and untwisting might be considered. At the age of seventy-eight an ovarian cyst, presumably malignant, might have perforated. Such a lesion could have twisted, become gangrenous and ruptured.

The vaginal discharge could be explained by the polyp in the cervix and also by the fact that the patient, who was seventy-eight years of age, had atrophy of the vagina and secretion from a moderate vaginitis. The fact that the vaginal smear was reported as negative means that probably there was not cancer of the endometrium. That is not a perfect method of diagnosis of endometrial cancer because in 20 per cent of cases the diagnosis is not made on the smear. The rather sudden onset of pain with fever makes one think of necrosis or infection. The fact that the x-ray department is willing to say that there is evidence of diverticulitis present makes one think that she must have had diverticulitis. Whether or not this is the whole answer to the problem I cannot say.

Was this mass movable, Dr. McKittrick?

Dr. McKittrick: No, it was not.

Dr. Meigs: If this was not a movable mass, I think that the best diagnosis is diverticulitis. On the other hand it might easily have been an ovarian cyst that was twisted or ruptured. One would think an ovarian cyst probably would be freely movable and more easily accessible than this seems to have been.

There is one other condition that I think one must consider, and that is old pelvic inflammation. In the past I have seen large, thick-walled pus tubes associated with a temperature of about this height and intermittent pain in the side. One does not often suspect that a patient seventy-eight years of age will have salpingitis, but I have seen it. However, on the evidence presented in this case, it does seem that one must make a diagnosis of diverticulitis.

Dr. Benjamin Castleman: Dr. McKittrick, would you tell us the opinion on the ward?

Dr. McKittrick: The patient had considerably more back pain radiating down her left thigh than is apparent from the record, which however should not mislead one. In any event our first consideration was diverticulitis, but we did not believe that we had sufficient confirmatory evidence to be at all sure of this diagnosis. The initial barium enema showed a fairly normal-looking bowel, with little or no narrowing and little change in the mucosal pattern, with the possible exception of the widening of mucosal folds described by Dr. McCort. The patient was acutely ill, and her story was consistent with, but certainly not typical of, diverticulitis.

Clinical Diagnosis

Inflammatory mass, left iliac fossa.
? Ovarian cyst with secondary infection.

Dr. Meigs's Diagnosis

Diverticulitis.

Anatomical Diagnosis

Chronic diverticulitis of sigmoid, with old abscess formation.

Pathological Discussion

Dr. McKittrick: We found a hard, fixed mass occupying the entire left iliac fossa. It is often true in diverticulitis, I think, that the surgeon finds more extensive alteration in the bowel at operation than seems likely by study of the barium-enema examination. The bowel wall is likely to be more indurated and thickened and difficult to handle. Changes in the bowel wall are very likely to be marked well above and below the site of perforation. This case was unusual in that the exposed and visible portion of sigmoid overlay the mass. The mass itself was in relation to the lateral wall of the sigmoid and was densely adherent to the posterior surface of the uterus and to the dome of the bladder. It was apparent that this was most likely an inflammatory process and most probably had arisen from the sigmoid, presumably from the perforation of a single diverticulum. It gave the impression of a very chronic process, with recently superimposed acute inflammatory reaction.

Dr. Castleman: Will you tell us what you did at the first operation?

Dr. McKittrick: At the first operation we simply explored her and did a transverse colostomy. Her temperature subsided, discomfort disappeared, and

she was sent home for a period of recuperation.

When she returned the mass was still present, a little smaller, but still very hard and fixed. The patient was very anxious to avoid a colostomy; otherwise at the second operation I think I might have done a permanent sigmoid-end colostomy because of the fixation of the process. The left ureter was intimately involved in the wall of the abscess by dense scar, and I could not free it and leave what I considered to be a viable organ. Consequently, it was transected and then the sigmoid resected. A very easy, low anastomosis was done, and the ureter implanted into the lower descending colon. The ureterointestinal anastomosis was done proximal to the intestinal anastomosis. This was necessary because there was not an adequate length of ureter to do this any lower. Two weeks after operation the patient was doing very well. She had excreted no urine by rectum, but did excrete it via the transverse colostomy.

Dr. Meigs: I think you had a good deal of courage to put the ureter in above the anastomosis. In a case with bilateral nephrostomies shown at grand rounds recently the ureter was anastomosed into the sigmoid, and after thirty-three days the patient was just beginning to urinate through the colostomy.

Dr. McKittrick: This woman had a transverse colostomy and has been excreting urine through the colostomy since operation.

Dr. Meigs: Then you should have transplanted below the anastomosis.

Dr. McKittrick: That could not be done because of the amount of ureter removed.

Dr. Meigs: I would not give up hope yet.

Dr. McKittrick: She has done very well.

Dr. Meigs: Are you going to close the colostomy?

Dr. McKittrick: Yes; the question is when to do it. Assuming that she continues to do well, I think the decision will have to be based on when we believe the ureterointestinal anastomosis is as well healed as one can expect it to be. I would estimate that an interval of somewhere between three and six weeks should suffice. By that time the inflammatory reaction should have subsided sufficiently to give the left kidney as good a chance of survival as one could expect.

Dr. Meigs: Does the barium enema now go through the anastomosis?

Dr. McCort: We used Umbrathor in place of barium. This is a commercial preparation of thorium dioxide diluted with an equal part of water.* We believed that if there was any leak around the anastomosis or any reflux into the ureter, it would be safer to use a preparation with a minimum of solid residue. On examination the opaque medium passed readily from the rectum to the site of the colostomy in the transverse colon. The anastomosis was intact, and there was no reflux into the ureter.

Dr. Meigs: Was the lumen patent?

Dr. McCort: The width of the lumen at the site of the anastomosis was almost equal to that of the normal bowel.

Dr. Meigs: I think that you should wait to close the colostomy until urine flows in the right direction. It will probably pass through the rectum when the inflammatory process leaves.

Dr. McKittrick: I am not sure that it is only the anastomosis that stops the urine, Dr. Meigs. The rectal sphincter may contribute to this.

Dr. Castleman: Just one of the diverticula was infected. All the others were perfectly smooth, and the bowel was freely movable except in the one spot to which the mass, about 9 cm. in diameter and composed of brawny infected material, was attached.

Addendum

The patient began passing urine by rectum three weeks after operation, and ten days later the transverse colostomy was closed. The mild left hydronephrosis, which was shown by intravenous urogram after the ureterocolic anastomosis, improved, and the patient left the hospital ten days after closure of the colostomy feeling very well.

*Shull, H. J., Schulz, M.D., and Jones, C. M. Roentgen examination of defunctioned colon after ileostomy, with special reference to safety and practical value of thorium dioxide as contrast medium. *Gastroenterology* **12**:1-9, 1949.

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ANNUAL MEETING OF THE MASSACHUSETTS MEDICAL SOCIETY, 1950

Like the perennial news pictures of the Easter parade that might almost have been taken from previous years' files, except for slight changes in fashion, the announcement of another annual meeting of the Massachusetts Medical Society could be but a stereotyped replica of many that have gone before.

It is, indeed, to be expected that the one hundred and sixty-ninth in this unbroken succession of annual events will, even aside from its announcement, have much in common with its predecessors. These points in common, however, will stem from the great traditions of the occasion and the reliability of the Committee on Arrangements in matters of good taste. The details of the meeting, aside from protocol, will be as up to date as antibiosis; as modern as televised surgery. It may be hoped that for many loyal and devoted fellows of the Society this will still be, so far as medical gatherings are concerned, the greatest show on earth.

The preliminary events on the program will be the meeting of the supervising censors in the afternoon of May 15, at the Hotel Statler in Boston, followed by the Cotting Supper for councilors and the meeting of the Council that evening. Many weighty matters will be discussed. The next three days will be occupied with the general clinical sessions, morning and afternoon, and the special events that add flavor to the proceedings. On Tuesday morning, May 16, at 11 a.m., after the business of the annual meeting of the Society has been transacted, awards will be presented, and the annual oration will be delivered by Dr. John W. O'Meara, chief of the Department of Orthopedic Surgery of the Worcester City Hospital and orthopedic surgeon to St. Vincent Hospital, on "Safe Management of Fractures." The oration will be followed by the annual luncheon.

On Wednesday morning, May 17, specifically at 11:05, Dr. Merrill C. Sosman, professor of radiology at the Harvard Medical School and radiologist-in-chief of the Peter Bent Brigham Hospital, will deliver the time-honored Shattuck Lecture, a matter of the utmost gravity, on "The Specificity and Reliability of Roentgen Diagnosis."

Fifty-five minutes later the various special sections will assemble separately for luncheon and other, sterner, pabulum; three new sections, that of Psychiatry and Neurology, that of Ophthalmology and Otolaryngology and that of Industrial Health being added as newcomers to the list. At the annual dinner on Wednesday evening the guest speaker will be Claude M. Fuess, Ph.D., Litt.D. (hon.), former headmaster of Phillips Andover Academy. On this occasion the new Massachusetts Medical Society Citation for Outstanding Contributions to Medical Progress will be awarded to its first recipient, Thomas F. Costello, editor and publisher of *The Lowell Sun*.

Concurrently with the meetings of the House of Lords, the Woman's Auxiliary, considerably fairer and no less effective, will hold its sessions on May 16 and 17. On Tuesday afternoon in the *Salle Moderne* — appropriately enough — Mrs. Frank Mansfield

Taylor, the well known lecturer, will speak on "Our World This Afternoon." On the following day there will be a bus tour and a luncheon, after which the two programs will unite for the evening.

The Massachusetts Medico-Legal Society will hold its meeting in the afternoon of May 17, and the exhibition of works of art by members of the Massachusetts Physicians' Art Association will be open daily from 9 to 5. The annual golf tournament will be held in the afternoon of May 17 at the Dedham Country and Polo Club, and the fellows are urged at all times to Visit the Exhibits!

But the complete program, after all, will be found on pages 726-732, and it is expected that a good time will be had by all.

THE SHATTUCK REPORT

THE current year is memorable as the centenary of an epoch-making work in public health in Massachusetts: the publication in 1850 of a sanitary survey of the Commonwealth by a special commission of three members authorized by the Legislature and appointed by Governor Briggs.

The report of 541 pages was entitled *Report of a General Plan for the Promotion of Public and Personal Health, Devised, Prepared and Recommended by the Commissioners Appointed under a Resolve of the Legislature of Massachusetts Relating to a Sanitary Survey of the State.* The main body of the report is divided into four parts: sanitary movement abroad; sanitary movement at home; plan for a sanitary survey of the State, with measures constituting the plan; and reasons for approving the plan recommended (including answers to objections and an appeal for the plan). A bill recommended for enactment is attached. An appendix of 219 pages presents the documentary evidence upon which the report is founded. The plan embodied fifty essential points, and it is interesting to note that almost all these items have become part of the laws of Massachusetts. Likewise, the bill recommended eventually led to the establishment of the Board of Health twenty years later.

The Commission before beginning work defined the subject under consideration as follows:

> By a sanitary survey of the State, is meant, an examination or survey of the different parts of the Commonwealth, — its counties, its towns, and its localities, — to ascertain the causes which favorably or unfavorably affect the health of its inhabitants. The word sanitary means relating to health. When we speak of the sanitary condition of a town, we include a description of those circumstances which relate to, or have an effect upon, the health of its inhabitants. When applied to the inhabitants of a town or district in their social capacity, it relates to public health; when to individuals, it relates to personal or private health.

The enunciation of this policy and the recommendation of a comprehensive plan placed Shattuck years ahead of the times in the field of public health.

Lemuel Shattuck, 1793-1879, layman, statistician and genealogist, was the prime mover of the project, and the author of the report. He was always interested in public affairs. He became a member of the Legislature in 1838 and set up business as a publisher and bookseller in Boston in 1840. He retired from business at the age of forty-six and devoted the remainder of his life to public service. In studying local genealogy he found that vital statistics had been greatly neglected, and he joined with others in 1839 in forming the American Statistical Association and, not long afterward, the New England Historic and Genealogical Society. He obtained the co-operation of the Massachusetts Medical Society and the American Academy of Arts and Sciences and was instrumental in obtaining the passage in 1842 of an act requiring the registration of births, marriages and deaths in the Commonwealth. In 1849, as a member of the Legislature, he was appointed chairman of a special committee on registration, leading to a thorough revision of the laws on registration. Shattuck, desiring more statistical information for his studies, persuaded the Boston Common Council, of which he had been an active member in 1837-1841, to take a census of the City in 1845. He was chosen to execute the project. His method of procedure made this census primarily one of persons rather than of families. For the first time in the United States, the data included the name and description and place of birth of every person enumerated. Shattuck also wrote an interpretative introduction to the report, an innovation that later became standard practice. The United States census of 1840

had been widely criticized, and when the time came for the national census of 1850, Shattuck was called to Washington as a consultant. He persuaded the Washington organizers to adopt many of the Boston procedures, which resulted in a much improved United States Census of 1850. Lemuel Shattuck is justly recognized as an outstanding pioneer in the field of public health. The original report, of which the first edition was issued in paper covers, bearing the title *Report of the Sanitary Commission of Massachusetts, 1850*, is a classic in its field and now much sought after by collectors, since only two thousand copies were ordered to be printed.

It is interesting to note that the Commission was appointed on July 3, 1849, and submitted its outstanding report to the Legislature on April 25, 1850 — a period of less than ten months for a large, special project, speaking well for the knowledge and indefatigability of Shattuck. A reprint of the report was made in 1948, but unfortunately all the appendix with its local surveys and reports was omitted. Professor C.-E. A. Winslow wrote in a preface to the reprint:

> Surely, this is an outstanding document for the year 1850 and it has its message for us in 1948. Johann Peter Frank, Chadwick, Simon, Sedgwick, Chapin, have written classics in the sciences of public health; but I know of no single document in the history of that science quite so remarkable in its clarity and completeness and in its vision of the future.

AESCULAPIAN WALKOUT?

To HELP resolve the suspicion that British physicians might invoke the strike as a protest against an unsatisfactory pay scale, reference to the *Medical World Newsletter* of the Medical Practitioners' Union for February and March may be of value.

The article having to do with trade unionism is entitled "Co-operating with the Inevitable." The question of striking is not raised, but the need is emphasized of a bargaining force, under present conditions, with more power than the average general practitioner standing alone possesses.

Such power is particularly necessary in Great Britain, according to the *Newsletter*, with private practice largely departed and nearly all physicians in the employ of the state. An agency for collective planning and collective bargaining, a thing not to be thought of forty years ago when practice was independent and a medical organization existed only for the exchange of clinical ideas, became essential with the introduction first of the panel system and more recently of the National Health Service.

Ministers will listen to a trade union with "the gigantic backing of the Trades Union Congress," who "are apt to be quite unconcerned with the justice of a claim if it comes from an average G. P. trying to fight a lone battle with his own limited weapons." The Medical Practitioners' Union thus justifies its own existence, with the added intimation that the British Medical Association cannot and should not, and in any event does not, adequately represent the profession in social and economic matters.

The possibility of even a limited walkout of physicians in Great Britain seems paradoxical enough, therefore, when raised, not by the obvious Medical Practitioners' Union, but by the Harrow Division of the British Medical Association. This Division, as reported in the Supplement to the *British Medical Journal* on March 11, passed the following resolution by a large majority:

> We, the members of the Harrow Area, are thoroughly dissatisfied with the low capitation fee paid to general practitioners under the National Health Service, and we hereby give notice that unless this is rectified by July, 1950, direct action will be taken by general practitioners in the Harrow Area to withhold our services under the terms of our contract, except for necessary medical attention.

British practitioners may be "Co-operating with the Inevitable," as the *Medical World Newsletter* has it. It seems probable, however, despite the joyous tidings that are brought to America by the southpaw division of the hit-and-run observers, that the life of all British physicians is not necessarily that of Reilly.

Strikes of physicians seem hardly consistent with the dignity and the high ethics of their calling. It must nevertheless be admitted that conditions giving rise to such ultimatums are not those that are likely to promote the continuance of these qualities. The services of the skilled physician of good repute have come to be considered as almost indispensable in the more advanced societies of the world. Such services may be withheld or lost entirely through forces more inexorable than a voluntary strike.

A CO-OPERATIVE ENTERPRISE

THE *Journal*, in its continued growth, remains mindful of the fact that it has now an average of some 20,000 subscribers to serve, in addition to the fellowship of the Massachusetts Medical Society. Of these 20,000 subscribers, well over 4500 are medical students who receive their subscriptions at considerably below cost. This deficit the *Journal* is willing to meet in the belief that it is performing some service in education, and that the difference will continue to be made up by the larger returns from regular subscribers and from the advertisers.

Obviously, the value of any publication as an advertising medium is directly dependent on the size of its bona-fide circulation and on that less tangible but equally important factor of the attention that the readers pay to the advertisements.

A publication actually sells two services, which are to a considerable degree interdependent. It sells its literary content to its readers, at a price that can be kept within reasonable bounds only by virtue of the income received from its advertisers, and it sells space to its advertisers only because of the number of readers before whom the advertisements will be displayed.

Your *Journal* — if a certain proprietary interest may be attributed to the subscribers — has been in the running for a hundred and twenty-two years. During that period, despite a normal experience of ups and downs, it has never been subsidized by the Society for a per-capita sum that has amounted to as much as the cost of its subscriptions.

This state of independent solvency will continue as long as its advertisers consider it a suitable medium for their purposes — a happy condition that will prevail as long as the readers co-operate in patronizing the advertisers and mentioning their *Journal* in so doing!

When the aid of the physician or surgeon is required to alleviate suffering, or save life, his services are duly appreciated; whereas when such aid is not in requisition, he often receives neglect and abuse.

Boston M. & S. J., *May 1, 1850*

BOSTON MEDICAL LIBRARY

CLASSIFICATION

On March 20, order number 600 was received for the third edition of the classification of the Boston Medical Library. This copy went to Cairo, Egypt. The system is used in a number of countries throughout the world and has been translated into Japanese and Spanish.

First published in the *Bulletin of the Medical Library Association*, in 1918, the classification was expanded, and a second edition published in 1925. Upon the exhaustion of this edition, a third was published in 1944 and 1946, in two parts: schedules, the classification proper, and a large assigning index for the complete schedules. This was the first published index, although the Library had a large manuscript index for the use of cataloguers. The third edition also included a condensed schedule for small libraries. These improvements enhanced the popularity of the classification, and it is now feared that the edition will soon be out of print again, since orders continue to come in steadily.

The original schedules were evolved in 1879 by Dr. James R. Chadwick, librarian, and Mrs. Elizabeth J. Collins, cataloguer, and were based on the *Index Catalogue of the Surgeon General's Office of the United States Army*. About 1915 Mr. Ballard began to revise the schedules to conform to the advances in modern medicine, and to set up a fundamental structure for future use, susceptible of expansion and revision, without altering the basic structure. It was also realized that a medical classification for libraries and others was to be used for assigning books to particular subjects and locations, and should not be expanded into an unworkable classification of knowledge. The alteration of a basic scheme entails enormous work upon library personnel in making changes and moving books to new locations, if the location is available. The classification is elastic, permitting expansion within any given subject without dismemberment of the fundamental structure. Mr. Ballard has been responsible for all the printed editions, and in the thirty-five years' experience of the Boston Medical Library the new scheme has proved satisfactory and easily handled.

OFFICERS OF THE MASSACHUSETTS MEDICAL SOCIETY, 1949-1950

Joseph Branzetti

DR. LELAND S. MCKITTRICK, *President-Elect*

PROGRAM OF THE ONE HUNDRED AND SIXTY-NINTH ANNIVERSARY OF THE MASSACHUSETTS MEDICAL SOCIETY

Monday, Tuesday, Wednesday and Thursday, May 15, 16, 17 and 18, Hotel Statler, Boston

The Registration Desk will be located on the mezzanine floor, and all who attend the meeting are requested to register.

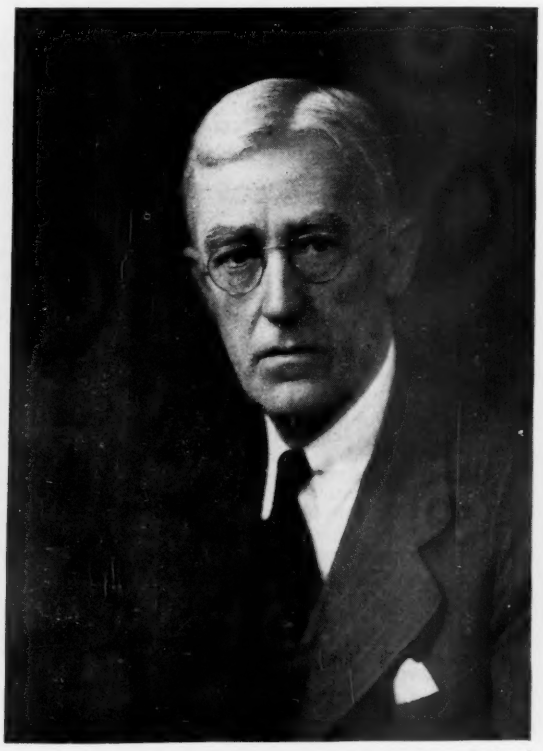
MONDAY AFTERNOON, MAY 15

4:30 **Supervising Censors' Meeting** (Parlor B).

6:00 **Cotting Supper for Councilors** (Parlors A and B).

White Star

Dr. Arthur W. Allen, *President*



Backrach

Dr. Albert A. Hornor, *Vice-President*

MONDAY EVENING, MAY 15

7:00 **Annual Meeting of the Council** (Georgian Room).

TUESDAY MORNING, MAY 16

First General Session

Georgian Room

Dr. Daniel B. Reardon, *Chairman*

Dr. John W. Spellman, *Co-chairman*

9:00 *A Motion Picture Entitled "Diagnostic Tests in Peripheral Arterial Disease."*

9:30 *Medical Malpractice.* Dr. Richard Ford, Boston. Assistant professor of legal medicine and acting head, Department of Legal Medicine, Harvard Medical School; associate medical examiner for Suffolk County.

9:55 *Difficulties in the Diagnosis of Obscure Fevers.* Dr. John Meyers, Worcester. Physician, Fallon Clinic and St. Vincent Hospital. Dr. James T. Brosnan, Worcester. Physician, Fallon Clinic and St. Vincent Hospital.

10:20 *Epidemic Poliomyelitis.* Dr. Harry M. Weaver, New York City. Director of Research, National Foundation for Infantile Paralysis, Inc.

11:00 **Annual Meeting of the Massachusetts Medical Society** (Georgian Room).

Presentation of Awards.

Annual Oration (following annual meeting): Safe Management of Fractures. Dr. John W. O'Meara, Worcester. Orthopedic surgeon-in-chief, Worcester City Hospital; orthopedic surgeon, St. Vincent Hospital.

Annual Luncheon (following annual oration) (PARLORS A AND B): tickets must be procured in advance of the meeting.

TUESDAY AFTERNOON, MAY 16

Second General Session

GEORGIAN ROOM

DR. ALBERT A. HORNOR, *Chairman*

DR. JOHN HOMANS, *Co-chairman*

2:00 *Clinical Uses of Estrogenic and Androgenic Compounds.* DR. LEWIS M. HURXTHAL, Boston. Head, Department of Internal Medicine, Lahey Clinic; physician, New England Deaconess and New England Baptist hospitals.

2:25 *Rehabilitation of Hemiplegics.* DR. HOWARD A. RUSK, New York City. Professor and chairman, Department of Physical Medicine and Rehabilitation, New York University College of Medicine; chief, Rehabilitation Service, Bellevue Hospital; director, Institute of Physical Medicine and Rehabilitation, New York University–Bellevue Medical Center.

2:50 *The Role of the Nonteaching Hospital in Medical Education.* DR. JAMES A. HALSTED, Jamaica Plain, Massachusetts. Chief, Medical Service, Faulkner Hospital.

Symposium on Peripheral Vascular Diseases

3:15 *Sympathectomy for Arteriosclerosis of the Lower Extremities.* DR. EDWARD A. EDWARDS, Boston. Clinical associate in anatomy, Harvard Medical School; associate in surgery, Peter Bent Brigham Hospital. DR. CHILTON CRANE, Boston. Instructor in surgery, Harvard Medical School; junior associate in surgery, Peter Bent Brigham Hospital.

3:40 *The Treatment of Varicose Veins.* DR. DAVID W. BARROW, Lexington, Kentucky. Surgeon-in-chief, Eastern Kentucky State Hospital; chief-of-staff, Stumbo Memorial Hospital; attending surgeon, St. Joseph's and Good Samaritan hospitals.

4:05 *Raynaud's Disease.* DR. ROBERT R. LINTON, Boston. Assistant professor of clinical surgery, Harvard Medical School; visiting surgeon, Massachusetts General Hospital; chief, Peripheral Vascular Clinic, Massachusetts General Hospital.

4:30 *A Motion Picture Entitled "Ureterosigmoidostomy for Exstrophy of the Bladder."* CAPTAIN HENRY W. HUDSON (MCR), USNR (Retired), U. S. Naval Hospital, Chelsea, Massachusetts.

TUESDAY EVENING, MAY 16

GEORGIAN ROOM

8:00 **Symposium on Nutrition in the Surgical Patient** (Presented by the Committee on Nutrition of the American College of Surgeons under the sponsorship of the Boston Surgical Society and the Massachusetts Medical Society.)

Some Biologic and Economic Factors in the Administration of Vitamins to Surgical Patients. DR. FREDRICK J. STARE, Boston. Professor of nutrition, Harvard School of Public Health; associate in medicine, Peter Bent Brigham Hospital.

Some Studies on the Nutritional Defects Found After Subtotal Gastrectomy. DR. PERRY J. CULVER, Boston. Associate in medicine, Harvard Medical School; assistant physician, Massachusetts General Hospital.

The Administration and Utilization of Amino Acids and Protein Hydrolysates. DR. CHARLES S. DAVIDSON, Boston. Assistant professor of medicine, Harvard Medical School; associate physician, Thorndike Memorial Laboratory, Boston City Hospital.

The Relation of Caloric Nourishment to Nitrogen Balance in the Postoperative Period. DR. HENRY T. RANDALL, New York City. Instructor in surgery, College of Physicians and Surgeons, Columbia University; assistant attending surgeon, Presbyterian Hospital.

Sodium and Potassium Requirements in the Surgical Patient as Related to Adrenocortical Function. DR. FRANCIS D. MOORE, Boston. Moseley Professor of Surgery, Harvard Medical School; surgeon-in-chief, Peter Bent Brigham Hospital.

WEDNESDAY MORNING, MAY 17

Third General Session

GEORGIAN ROOM

DR. PEIRCE H. LEAVITT, *Chairman*

DR. JAMES M. FAULKNER, *Co-chairman*

9:00 *A Motion Picture Entitled "We Speak Again — The Rehabilitation of Laryngectomized Patients."* DR. LEROY A. SCHALL, Massachusetts Eye and Ear Infirmary.

9:25 *The Health Protection Clinic.* DR. VLADO A. GETTING, Boston. Commissioner, Department of Public Health, Commonwealth of Massachusetts. DR. CLAIRE F. RYDER, Boston. Administrator, Health Protection Clinic, Department of Public Health, Commonwealth of Massachusetts.

9:50 *Deafness: Its practical aspects.* DR. DONALD K. LEWIS, Boston. Associate director, Winthrop Foundation, Massachusetts Eye and Ear Infirmary; assistant surgeon, Massachusetts Eye and Ear Infirmary and Massachusetts General Hospital. DR. PHILIP E. MELTZER, Boston. Surgeon-in-charge, Winthrop Foundation, Massachusetts Eye and Ear Infirmary.

10:15 *One Thousand Duodenal-Ulcer Patients: Results with and without surgery.* DR. FRANCIS D. MOORE, Boston. Moseley Professor of Surgery, Harvard Medical School; surgeon-in-chief, Peter Bent Brigham Hospital. In collaboration with DRS. W. P. J. PEETE, J. M. ERSKINE, J. R. BROOKS, JR., E. E. RICHARDSON and H. ROGERS.

10:40 *Results of the Boston Chest X-Ray Survey.* DR. JOHN H. CAULEY, M. P. H., Boston. Commissioner, Health Department, City of Boston.

11:05 **The Shattuck Lecture.** The Specificity and Reliability of Roentgen Diagnosis.

DR. MERRILL C. SOSMAN, Boston. Professor of radiology, Harvard Medical School; radiologist-in-chief, Peter Bent Brigham Hospital.

WEDNESDAY NOON — MAY 17

Section Meetings and Luncheons

12:00 m. — 2:00 p.m.

Section of Medicine

PARLOR A

DR. ALLEN S. JOHNSON, Longmeadow, *Chairman*

DR. JAMES A. HALSTED, Dedham, *Vice-chairman*

DR. FRANK B. CARR, Worcester, *Secretary*

Abuses of Antibiotic Therapy. DR. MAXWELL FINLAND, Boston. Associate professor of medicine, Harvard Medical School; associate physician, Thorndike Memorial Laboratory, and chief, Fourth Medical Service, Boston City Hospital.

Section of Pediatrics

PARLOR B

DR. STEWART H. CLIFFORD, Brookline, *Chairman*
DR. GERALD N. HOEFFEL, Boston, *Secretary*

Present-Day Ideas About Immunization. DR. R. CANNON ELEY, Boston. Associate in pediatrics and communicable diseases, Harvard Medical School; chief, Isolation Service, Infants' and Children's Hospital.

Section of Radiology

PARLOR C

DR. LAURENCE L. ROBBINS, Winchester, *Chairman*
DR. MAGNUS I. SMEDAL, Waban, *Secretary*

Developments in Cardiovascular and Cerebral Angiography. DR. WENDELL G. SCOTT, St. Louis, Missouri. Associate professor of clinical radiology, Washington University School of Medicine; reserve consultant in radiology, Bureau of Medicine and Surgery, Department of the Navy.

Section of Physical Medicine

HANCOCK ROOM

DR. SIDNEY LICHT, Cambridge, *Chairman*
DR. ARTHUR L. WATKINS, Arlington, *Secretary*

A Technic for the Recording of Posture. DR. WILLIAM BIERMAN, New York City. Attending physician in physical medicine, Mount Sinai Hospital.

Section of Ophthalmology and Otolaryngology

FOURTH FLOOR

DR. JAMES J. REGAN, South Boston, *Chairman*
DR. LAWRENCE R. DAME, Greenfield, *Secretary*

The Purpose of the New Section of Ophthalmology and Otolaryngology. DR. WALTER B. LANCASTER, Boston. Consulting ophthalmic surgeon, Massachusetts Eye and Ear Infirmary and Boston City Hospital.

Section of Industrial Health

FOURTH FLOOR

DR. DANIEL L. LYNCH, Roslindale, *Chairman*
DR. ALBERT O. SEELER, Waban, *Secretary*

Trends and Opportunities in Industrial Medicine. DR. EDWARD C. HOLMBLAD, Chicago. Senior attending surgeon, St. Luke's Hospital; managing director, American Association of Industrial Physicians and Surgeons.

WEDNESDAY AFTERNOON, MAY 17

Fourth General Session

GEORGIAN ROOM

DR. GRANTLEY W. TAYLOR, *Chairman*
DR. W. FENN HOYT, *Co-chairman*

2:00 *Newer Occupational Diseases.* MR. JOHN B. SKINNER, director, Division of Occupational Hygiene, Massachusetts Department of Labor and Industry.

2:25 *Farm Injuries.* DR. JOHN H. POWERS, Cooperstown, New York. Associate surgeon, Mary Imogene Bassett Hospital.

2:50 *Physical Medicine in the Treatment of Peripheral Nerve Injuries.* DR. WILLIAM BIERMAN, New York City. Attending physician in physical medicine, Mount Sinai Hospital.

3:15—*The Importance of Recognizing Early Cancer of the Colon.* DR. WENDELL G. SCOTT, St. Louis, Missouri. Associate professor of clinical radiology, Washington University School of Medicine; reserve consultant in radiology, Bureau of Medicine and Surgery, Department of the Navy.

What About Medical Laboratory Technologists?

3:40 *Moderator*: DR. ROBERT A. MACCREADY, Boston. Assistant director, Division of Communicable Diseases, Massachusetts Department of Public Health.

(A) *As Seen by the Educator.* PHILIP M. RICHARDSON, PH.D., Boston. Professor of biology, Simmons College.

(B) *As Seen by the Technologist.* MRS. ELINOR JUDD, B.A., M.T.(ASCP), chief medical technologist, Robert Breck Brigham Hospital.

(C) *As Seen by the Pathologist.* DR. DONALD A. NICKERSON, Melrose, Massachusetts. Associate professor of pathology, Boston University School of Medicine; pathologist, Salem Hospital, Salem, Massachusetts.

4:30 *A Motion Picture Entitled "Examination of the Breast for Early Cancer."*

WEDNESDAY EVENING, MAY 17

6:00–7:00 **Cocktail Party** (SALLE MODERNE).

Ladies may be invited.

7:00 **Annual Dinner of the Massachusetts Medical Society** (GEORGIAN ROOM).

Presiding:
DR. ARTHUR W. ALLEN, president, Massachusetts Medical Society.

Guest Speaker:
CLAUDE M. FUESS, PH.D., LITT.D. (hon.).

Presentation of Award to:
THOMAS F. COSTELLO, editor and publisher, *The Lowell Sun*, Lowell, Massachusetts

Ladies may be invited to attend the dinner.
Tickets for the dinner *must* be procured in advance.

THURSDAY MORNING, MAY 18

Fifth General Session

GEORGIAN ROOM

DR. CURTIS C. TRIPP, *Chairman*
DR. CHARLES G. SHEDD, *Co-chairman*

9:00 *A Motion Picture Entitled "Diseases of the Gall Bladder."*

9:25 *Management of the Neurotic Patient.* DR. DONALD J. MACPHERSON, Boston. Senior associate in psychiatry, Peter Bent Brigham Hospital.

9:50 *Rehabilitation of War Wounds of the Hand.* DR. RICHARD WARREN, West Roxbury, Massachusetts. Consultant in surgery, Veterans Administration Hospital. DR. D. RICHARD FRENI, West Roxbury, Massachusetts. Attending surgeon, Veterans Administration Hospital.

10:15 *Cesarean Section with All Its Present-Day Concepts.* DR. MARJORIE WOODMAN, Boston. Chief, Obstetrical Service, New England Hospital for Women and Children.

10:40 *Management of Head Trauma.* DR. JEFFERSON BROWDER, Brooklyn, New York. Professor of surgery, The State University Medical Center at New York.

11:05 *The Treatment of Acute Infectious Diseases with the Newer Antibiotics.* Dr. Theodore E. Woodward, Baltimore, Maryland. Associate professor of medicine, University of Maryland School of Medicine.

11:30 *Virus Pneumonia.* Dr. Lowrey F. Davenport, Boston. Physician, Boston Dispensary; assistant in medicine, Massachusetts General Hospital.

THURSDAY NOON, MAY 18

Section Meetings and Luncheons

12:00 m. — 2:00 p.m.

Section of Surgery

Salle Moderne

Dr. Franklin G. Balch, Jr., Chestnut Hill, *Chairman*

Dr. J. Hartwell Harrison, Brookline, *Secretary*

Neurosurgical Treatment of Intractable Pain. Dr. William H. Sweet, Boston. Assistant professor of surgery, Harvard Medical School; neurosurgeon-in-chief, New England Center Hospital; associate visiting neurosurgeon, Massachusetts General Hospital.

Section of Obstetrics and Gynecology

Parlor A

Dr. Daniel J. McSweeney, Milton, *Chairman*

Dr. Duncan E. Reid, Boston, *Vice-chairman*

Dr. Arthur J. Gorman, Newton, *Secretary*

Anti-Pitressin Factor in the Treatment of Dysmenorrhea and Menstrual Molimina. Dr. William Bickers, Burlington, Vermont. Chairman, Department of Obstetrics and Gynecology, Bishop DeGoesbriand Hospital.

Section of Dermatology and Syphilology

Parlor C

Dr. Maurice M. Tolman, Chelsea, *Chairman*

Dr. John Adams, Jr., Roxbury, *Secretary*

Tinea Capitis. Dr. Donald M. Pillsbury, Philadelphia, Pennsylvania. Professor and chief, Department of Dermatology and Syphilology, University of Pennsylvania School of Medicine.

Section of Anesthesiology

Parlor B

Dr. Jacob Fine, Beverly, *Chairman*

Dr. Leo V. Hand, Newton Highlands, *Secretary*

Metabolic Fate of Pentothal Sodium in Man. Dr. Emanuel M. Papper, New York City. Professor of anesthesiology, Columbia University College of Physicians and Surgeons; director, Department of Anesthesia, Presbyterian Hospital.

Section of Physiology and Pathology

Fourth Floor

Dr. Donald A. Nickerson, Melrose, *Chairman*

Dr. Stanley L. Robbins, Brookline, *Secretary*

Newer Aspects of the Pathology of Acute Lupus Erythematosus. Dr. Paul Klemperer, New York City. Pathologist, Mount Sinai Hospital.

Section of Psychiatry and Neurology

Fourth Floor

Dr. Walter E. Barton, Dorchester, *Chairman*

Dr. Augustus S. Rose, Belmont, *Secretary*

Emotions and Clinical Medicine. Dr. Stanley Cobb, Boston. Bullard Professor of Neuropathology, Harvard Medical School; psychiatrist-in-chief, Massachusetts General Hospital.

THURSDAY AFTERNOON, MAY 18

Sixth General Session

Georgian Room

Dr. W. Richard Ohler, *Chairman*

Dr. Allen S. Johnson, *Co-chairman*

2:00 *The Anesthesiologist as a Practitioner of Medicine in the Community.* Dr. Emanuel M. Papper, New York City. Professor of anesthesiology, Columbia University College of Physicians and Surgeons; director, Department of Anesthesia, Presbyterian Hospital.

2:25 *The Management of Sterility.* Dr. Edmond J. Farris, Philadelphia, Pennsylvania. Executive director, Wistar Institute of Anatomy and Biology.

2:50 *The Management of Chronic Dermatitis.* Dr. Donald M. Pillsbury, Philadelphia, Pennsylvania. Professor, Department of Dermatology and Syphilology, University of Pennsylvania School of Medicine.

3:15 *Cortisone and ACTH: Clinical Aspects in Rheumatoid Arthritis.* Dr. J. Sydney Stillman, Boston. Chief, Medical Service, Robert Breck Brigham Hospital.

3:40 *Cortisone and ACTH: Metabolic Effects.* Dr. George W. Thorn, Boston. Hersey Professor of the Theory and Practice of Physic, Harvard Medical School; physician-in-chief, Peter Bent Brigham Hospital.

4:05 *Post-Cholecystectomy Symptoms.* Dr. S. Allen Wilkinson, Boston. Department of Gastroenterology, Lahey Clinic.

4:30 *A Motion Picture Entitled "Crushing Injuries of the Thorax."*

SCIENTIFIC EXHIBITS

BOOTH

81 — Principles in the Preparation of Flaps.
Exhibitor: Bradford Cannon, M.D.

82 — Contributions of Cytologic Methods.
Sponsor: Harvard Cancer Commission, Laboratory of Pathology.
Exhibitors: Shields Warren, M.D., and Olive Gates, M.D.

83 — The Surgery of Complicated Duodenal Ulcer in a Veterans Administration Hospital.
Sponsor: Veterans Administration Hospital, West Roxbury.
Exhibitors: Frederick P. Ross, M.D., Richard Warren, M.D., Edmund C. Meadows, M.D., and Robert H. Vaughan, M.D.

84 — Organization and Activities for Industrial Health.
Sponsor: Section of Industrial Health.

85 — Treatment in the State Mental Hospitals.
Sponsor: Boston State Hospital.
Exhibitor: Section of Neurology and Psychiatry.

86 — Orthopedic Series Illustrating the Bankart Operation for Recurrent Dislocation of the Shoulder.
Sponsor: United States Naval Hospital, Chelsea, Massachusetts.
Exhibitor: Lieutenant Commander William C. Roland (MC), USN.

87 — The Handicapped in the Process of Rehabilitation.
Sponsor: Community Workshops, Boston.
Exhibitors: Staff members of the Community Workshops.

88 — Rheumatoid Arthritis.
Sponsor: Robert Breck Brigham Hospital.
Exhibitors: Theodore A. Potter, M.D., and Théodore B. Bayles, M.D.

89 — The Health-Protection Clinic.
Sponsor: Massachusetts Department of Public Health.
Exhibitors: Vlado A. Getting, M.D., Claire F. Ryder, M.D., and Herbert L. Lombard, M.D.

90 — Transplantation of the Cornea.
Exhibitor: Brendan D. Leahey, M.D.

91 — Medical Mycology.
Sponsor: Harvard Medical School.
Exhibitors: Jacob H. Swartz, M.D., Ruth Cutter Burke, Ph.D., and Earl A. Glicklich, M.D.

92 — New Concepts of the Etiology and Treatment of Hirschsprung's Disease.
Sponsor: The Children's Hospital.
Exhibitors: Orvar Swenson, M.D., and Edward B. Neuhauser, M.D.

TECHNICAL EXHIBITORS
1950

	BOOTH NO.
Abbott Laboratories	56
Alkalol Company	22
Ames Company, Inc.	42
Atlantic X-Ray Company	32
Ayerst, McKenna & Harrison, Ltd.	19
Baker Laboratories	31
Chester A. Baker Laboratories, Inc.	78
Beech-Nut Packing Company	76
Bilhuber-Knoll Corporation	34
The Borden Company	33
Brewer & Company, Inc.	4 & 5
Brown and Connolly, Inc.	57-A
Buffington's	41
Burroughs Wellcome & Company, Inc.	24
Cambridge Instrument Company, Inc.	79
Camel Cigarettes	11 & 12
Carnation Company	6
Ciba Pharmaceutical Products, Inc.	39
Coca-Cola Bottling Company of Boston	13 & 14
Crosbie-Macdonald	46
Davies, Rose & Company, Ltd.	40
Denver Chemical Manufacturing Company, Inc.	73
Doho Chemical Corporation	58
Dy-Dee Service, Inc.	10
Electro-Physical Laboratories	7-A
Electronic Surgical Equipment Company, Inc.	54
Endo Products, Inc.	61
C. B. Fleet Company, Inc.	43
General Electric X-Ray Corporation	47 & 48
Gerber's Baby Foods	80
J. E. Hanger, Inc.	35
Hanovia Chemical & Manufacturing Company	52
Hoffmann–LaRoche, Inc.	29
H. P. Hood & Sons	60
Kelley–Koett X-Ray Company	1
George Laben	30
Lea & Febiger	3
Lederle Laboratories Division	74
Eli Lilly and Company	17
J. B. Lippincott Company	7
M & R Dietetic Laboratories, Inc.	16
E. F. Mahady Company	Stage A, B, C
Maltbie Chemical Company	8
McIntosh Electric Corporation	55
McNeil Laboratories, Inc.	77
Mead Johnson & Company	44 & 45
Medical Clearing Bureau, Inc.	59
Medical Protective Company	9
The Wm. S. Merrell Company	70
The National Drug Company	38
New England Dairy & Food Council	2
T. J. Noonan Company	25
Parke, Davis & Company	72
E. L. Patch Company	15
Pet Milk Company	75
Philip Morris & Company, Ltd., Inc.	49
Professional Equipment & Hospital Supply Company	57
L. & B. Reiner	20 & 21
Sanborn Company	27
Sandoz Chemical Works, Inc.	37
Saratoga Springs Authority	12-A
Schering Corporation	65
G. D. Searle & Company	71
Sharp & Dohme, Inc.	50
Smith, Kline & French Laboratories	36
South Shore Office Supply & Equipment Company	Stage D, E, F
Spencer, Inc.	62
E. R. Squibb & Sons	69
Surgeons' & Physicians' Supply Company	63
Tailby–Nason Company	18
The Upjohn Company	51
Vaponefrin Company	66
U. S. Vitamin Corporation	23
Westinghouse Electric Corporation	67 & 68
White Laboratories, Inc.	28
Winthrop–Stearns, Inc.	53
Wyeth, Inc.	64
F. E. Young & Company	26

MASSACHUSETTS MEDICO-LEGAL SOCIETY ANNUAL MEETING

WEDNESDAY AFTERNOON, MAY 17

PARLOR C

2:30 p.m.

EXHIBITION OF WORKS OF ART

by Members of the Massachusetts Physicians' Art Association

ROOM 419

Daily: 9:00 a.m. — 5:00 p.m.

MAY 16, 17 and 18

ANNUAL GOLF TOURNAMENT

DEDHAM COUNTRY AND POLO CLUB,

DEDHAM, MASSACHUSETTS

WEDNESDAY AFTERNOON — MAY 17

1 p.m.

DR. CHARLES G. SHEDD, *Chairman*

1180 Beacon Street

Brookline, Mass.

WOMAN'S AUXILIARY OF THE MASSACHUSETTS MEDICAL SOCIETY

The registration desk will be located on the mezzanine floor, Hotel Statler. Registration will be from 9:00 a.m. to 4:00 p.m. on both days of the meeting.

TUESDAY MORNING, MAY 16

11:00 **Annual Meeting** (Parlor B, Hotel Statler).

TUESDAY AFTERNOON, MAY 16

1:00 **Luncheon** (China House, 146 Boylston Street, Boston; $1.25, including Tax and Tip).

2:15 **Executive Meeting** (Parlor F, Hotel Statler).

3:00–5:00 **Lecture** (Salle Moderne, Hotel Statler). *Our World This Afternoon.* MRS. FRANK MANSFIELD TAYLOR.

WEDNESDAY MORNING, MAY 17

11:00 **Bus Tour Starting from Columbus Avenue Entrance of the Hotel Statler for the Cliff Hotel, North Scituate.** (Transportation provided.)

WEDNESDAY AFTERNOON, MAY 17

1:00 **Luncheon** (Cliff Hotel, North Scituate; $1.50, including Tax and Tip).

2:00–3:30 **Cards.**

WEDNESDAY EVENING, MAY 17

6:00–7:00 **Cocktail Party** (with members of Massachusetts Medical Society in Salle Moderne, Hotel Statler).

7:00 **Dinner** (with members of Massachusetts Medical Society in Georgian Room, Hotel Statler).

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

COMMUNICABLE DISEASES IN MASSACHUSETTS FOR MARCH, 1950

RÉSUMÉ

DISEASES	MARCH 1950	MARCH 1949	SEVEN-YEAR MEDIAN
Chancroid	2	7	3*
Chicken pox	2414	4026	2405
Diphtheria	18	39	21
Dog bite	822	946	790
Dysentery, bacillary	15	4	9
German measles	351	891	316
Gonorrhea	211	274	327
Granuloma inguinale	1	0	0*
Lymphogranuloma venereum	1	3	2*
Malaria	0	0	8
Measles	1529	6551	3546
Meningitis, meningococcal	9	7	12
Meningitis, Pfeiffer-bacillus	4	0	4
Meningitis, pneumococcal	2	3	2
Meningitis, staphylococcal	0	0	0
Meningitis, streptococcal	0	1	1
Meningitis, undetermined	9	3	5
Mumps	1343	1790	1575
Poliomyelitis	2	0	2
Salmonellosis	11	2	8
Scarlet fever	653	1212	1212
Syphilis	167	218	412
Tuberculosis, pulmonary	216	170	228
Tuberculosis, other forms	14	14	20
Typhoid fever	0	0	1
Undulant fever	1	7	5
Whooping cough	776	319	592

*Six-year median.

COMMENT

Diseases above the seven-year median during the month of March included dog bite, bacillary dysentery, German measles and whooping cough.

Diseases below the seven-year median were diphtheria, measles, mumps and scarlet fever.

Whooping cough (776 cases) reached the highest point for March since 1945, when 853 cases were reported. This was the third consecutive month in which whooping cough exceeded the seven-year median and the thirteenth month in which it exceeded last year's figures.

Measles has remained considerably below the seven-year median for the past twelve months, and scarlet fever below the seven-year median for the ninth month. Only three times since 1918 has March shown fewer cases of the latter disease.

Diphtheria was at the lowest level since 1943.

GEOGRAPHICAL DISTRIBUTION OF CERTAIN DISEASES

Anthrax was reported from: Peabody, 1; total, 1.

Diphtheria was reported from: Boston, 7; Chelsea, 3; Cohasset, 1; Danvers, 2; East Bridgewater, 1; Hingham, 1; New Bedford, 1; Revere, 1; Weymouth, 1; total, 18.

Dysentery, bacillary, was reported from: Belchertown, 6; Boston, 5; Waltham, 2; Worcester, 2; total, 15.

Encephalitis, infectious, was reported from: Worcester, 2; total, 2.

Hepatitis, infectious, was reported from: Wrentham, 2; total, 2.

Meningitis, meningococcal, was reported from: Boston, 2; Boxford, 1; Brockton, 2; Chelsea, 1; Lawrence, 1; Lowell, 1; Somerville, 1; total, 9.

Meningitis, Pfeiffer-bacillus, was reported from: Franklin, 2; Lynn, 1; South Hadley, 1; total, 4.

Meningitis, pneumococcal, was reported from: Norwood, 1; Worcester, 1; total, 2.

Meningitis, mumps, was reported from: Mansfield, 1; total, 1.

Meningo-encephalitis was reported from: Wakefield, 1; total, 1.

Meningitis, undetermined, was reported from: Amesbury, 1; Ashland, 1; Boston, 1; Peabody, 1; Pittsfield, 2; Rockland, 1; Springfield, 1; Wellesley, 1; total, 9.

Poliomyelitis was reported from: Deerfield, 1; Groton, 1; total, 2.

Salmonellosis was reported from: Boston, 2; Everett, 2; Gloucester, 1; Lexington, 1; Newton, 1; Quincy, 2; Salem, 1; Worcester, 1; total, 11.

Septic sore throat was reported from: Belmont, 1; Boston, 2; Fall River, 1; Lynn, 2; Malden, 1; Medford, 1; Templeton, 3; Worcester, 2; total, 13.

Undulant fever was reported from: Brockton, 1; total, 1.

CORRESPONDENCE

X-RAY SURVEY FOLLOW-UP EXAMINATIONS

To the Editor: During the course of the Boston Chest X-Ray Program, a number of persons whose chest films showed suspicion of cancer of the lung were advised to consult their own physicians for further study of their chest conditions.

The Committee on Nontuberculous Chest Diseases of the Boston Chest X-Ray Survey, consisting of Dr. Thomas J. Anglem, chairman, Dr. Herbert D. Adams, Dr. Theodore L. Badger, Dr. Howard A. Frank, Dr. Joseph Lynch, Dr. Harlan F. Newton, Dr. Richard H. Overholt, Dr. John W. Strieder and Dr. Richard H. Sweet, is anxious to complete these records and evaluate the data. It would be most helpful, therefore, if all physicians to whom survey findings suspicious of cancer have been sent would return the survey forms with their final diagnosis to Boston Chest X-Ray Headquarters, 57 East Concord Street.

If this is not convenient, the final diagnosis may be reported by telephone (LIberty 2-8814, Mrs Rudolf Osgood).

THOMAS J. ANGLEM, M.D., *Chairman*
Committee on Nontuberculous Chest Diseases
Boston Chest X-Ray Survey

ON THE RESIGNATION OF DR. PERKINS

To the Editor: May I commend you for your splendid editorial on the resignation of Dr. Clifton T. Perkins, which someone has been kind enough to forward to me?

Your vigilant interest in the problems of mental health can do a great deal to forward the work in which Dr. Perkins was engaged, and can help us achieve the standards for which he strived.

A majority of the "politicians" on Beacon Hill are as anxious as the medical profession to restore mental patients to a useful place in society. With your help, I hope that we can prevent this great work from being sabotaged further by those hungry for political patronage.

Good administrators must be allowed to administrate. Good mental health and good mental health programs are dependent on good (and alert) citizenship.

HAROLD PUTNAM
House of Representatives,
State House, Boston

REGARDING RADIOACTIVE COBALT

To the Editor: One of the signs of the times is the increasing frequency with which universities, hospitals and individuals present the advances and discoveries, alleged or otherwise, of medicine in the public press instead of in accepted medical journals, their rightful place.

A case in point is the recent newspaper publicity regarding radioactive cobalt. The radioactive isotopes, P32 and I-131, have established their right to a place in the diagnostic and therapeutic armamentarium. Cobalt 60 is another story. Very little clinical work has been done with it. No five-year end-results have been published, because there are none. This is not to say that cobalt 60 will not prove to be a very useful tool in radiation therapy, but to give the impression that one should sell one's radium at this time and buy radioactive cobalt is putting the cart before the horse.

On the Continent of Europe and in a few places in this country, radium packs of 2, 4 and 10 gm. have been used for external therapy. Such installations are expensive since radium ore is mined, refined and sold commercially. Much of the interest in cobalt 60 has been stimulated by the price at which it can be purchased through governmental subsidy, thus suggesting the installation of large packs whose radiation could be compared with multi-million-volt x-ray generators.

When it is stated that cobalt 60 can be purchased for one five-hundredth to one one-thousandth of the price of radium, we should remember that that is possible only when the cost of the atomic pile ($2,000,000,000), the cost of keeping the atomic pile operating (several million dollars a year) and 50 per cent of the actual handling costs for the preparation of this isotope are written off.

As a matter of fact, as far as radiation is concerned, it is almost impossible to distinguish between that from a 2,000-000 volt x-ray machine and that from the 1000-curie cobalt 60 pack, on the one hand; on the other, for small sources, the radiation is very closely the same as that from radium. Therefore, there is no new modality at all. It offers no substitute for radon. The inestimable value of the latter as gold implants can only be appreciated by the initiated.

Radium-element needles are a constant for all practical purposes and are as useful today as they ever were, which is directly proportional to the experience of the one using them.

Individual physicians and hospitals have purchased radium at a high price over the years and have a substantial interest in it.

It does not make sense to jeopardize these investments by ill advised statements or minimize the benefit accomplished by radium, so that physicians must apologetically explain to patients why they use radium instead of cobalt 60.

In the future, radium may be replaced by the development of new radioactive isotopes. What better peacetime use for the atomic pile? Meanwhile, the propaganda should be stopped until experience and data justify it.

FREDERICK W. O'BRIEN, M.D.
465 Beacon Street
Boston, Massachusetts

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

May's Manual of the Diseases of the Eye for Students and General Practitioners. Revised and edited by Charles A. Perera, M.D., assistant clinical professor, College of Physicians and Surgeons, Columbia University, and associate attending ophthalmologist, Presbyterian Hospital, New York City. Twentieth edition. 12°, cloth, 512 pp., with 378 illustrations. Baltimore: Williams and Wilkins Company, 1949. $5.00.

This manual, written primarily for medical students and general practitioners, was first published in 1900. It has an enviable publication record. In addition to the 20 American editions, there have been 10 German editions and translations into nine foreign languages, including Chinese, Japanese and Urdu. The Spanish translation is now in the eleventh edition. This last American edition has been completely revised and brought up to date, and the type reset. New and revised material includes the treatment of conjunctivitis, the classification and therapy of diseases of the optic nerve, the use of the newer antibiotics, the surgical correction of oculomotor abnormalities and the ocular manifestations of systemic diseases. The book is well published in every way, and its astounding popularity is indicative of the soundness of the text.

Golden Jubilee World Tribute to Dr. Sidney V. Haas: In honor of his pioneering contribution to celiac therapy and the treatment of the hypertonic infant, and of the completion of his fiftieth year of medical practice. 8°, cloth, 37 pp., illustrated. New York: The Committee for the Golden Jubilee Tribute to Dr. Sidney V. Haas, 1949.

This small volume constitutes a record of the tribute tendered to Dr. Haas at a luncheon held at the New York Academy of Medicine on April 5, 1949. A foreword by Dr. Evan Evans emphasizes the two outstanding therapeutic contributions of Dr. Haas to pediatrics: the use of bananas in the treatment of celiac disease, and atropin in the treatment of the hypertonic infant. There follows a short biography of Dr. Haas, and his speech made in acceptance of a *Golden Book of World Tributes*. Extracts from some of the letters, with a complete list of contributors, concludes the volume, which should be included in all collections on the history of medicine and should prove interesting to pediatricians.

Cancer: New light on its causes, detection, treatments, cure and the brilliant promise of today's research. By Beka Doherty. 8°, cloth, 327 pp. New York: Random House, 1949. $3.00.

This popular book on a subject of paramount interest is well written and covers the various aspects of cancer in relation to the patient and to the physician, and as a research and public problem. An important appendix gives lists of cancer clinics in approved hospitals, in the United States and Canada; approved detection centers; tissue diagnostic services; and foundations interested in cancer in the United States. These up-to-date lists should prove very valuable to medical-reference librarians. Miss Doherty had the privilege of using the facilities of the Memorial Hospital and the Sloan-Kettering Institute while writing her book. It should be in all medical and public libraries.

Cystoscopy and Urography. By James B. Macalpine, D.Sc. (Honoris Causa), F.R.C.S. (Eng.), consulting urologist, Christie Hospital and Holt Radium Institute, Manchester. Third edition. 8°, cloth, 554 pp., with 338 illustrations. Baltimore: Williams and Wilkins Company, 1949. $13.50.

The previous edition of this British book was published in 1936, and the complete stock of copies and all the material needed for a reprint were destroyed when the publishing house in London was bombed during the air raids. This third edition has been revised and brought up to date, and material has been added throughout the text. A new section on the punch operation for prostatic hypertrophy has been contributed by Mr. R. H. O. B. Robinson. The chapter on urinary tuberculosis has been largely rewritten and preceded by a chapter on the pathology of that disease. The chapter on bilharziasis has been much enlarged. There is a good index, and the publishing is good. The printing was done in Great Britain.

Microbiology. By Florence C. Kelly, M.S., Ph.D., associate professor of bacteriology, University of Oklahoma School of Medicine; and K. Eileen Hite, Ph.D., M.D. With an introduction by G. M. Dack, Ph.D., M.D., professor of bacteriology, University of Chicago School of Medicine. 8°, cloth, 592 pp., with 185 illustrations and 22 tables. New York: Appleton–Century–Crofts, Incorporated, 1949. $6.50.

This new book is intended for the student interested in the fields of general and pathogenic microbiology. The text divides naturally into two divisions. The first four parts deal with the biology and inter-relations of the large phylogenetic groups, bacterial physiology, microbial populations and their control, and methods for the study of micro-organisms. The succeeding parts discuss micro-organisms and disease; pathogenic microbiology, including the bacteria, viruses, the higher fungi and protozoa; and epidemiology and public health. An appendix comprises a list of early milestones in microbiology, beginning with Roger Bacon (1267) on optics and ending with Albert Calmette (1924) on antituberculosis vaccination in children. There is also a list of technical methods not already included in the text. The text is concluded with a bibliography of eighteen pages arranged by chapters. There is a good index, and the book is well published and should prove useful to students of the subject.

Physiology of the Uterus. By S. R. M. Reynolds, M.A., Ph.D., staff member and physiologist, Department of Embryology, Carnegie Institution of Washington, Baltimore, and lecturer on obstetrics, Johns Hopkins University School of Medicine, Baltimore. Second edition. 4°, cloth, 611 pp., with 67 illustrations. New York: Paul B. Hoeber, Incorporated, 1949. $12.50.

This standard reference work, first published in 1939, has been completely revised and considerably enlarged. The material has been reorganized into eight sections, containing closely related chapters. Material has been added on human tocography, especially that concerned with multichannel recording of patterns of uterine contractions; steroid hormones during pregnancy; factors in abnormal uterine growth; myometrial forces in relation to uterine growth and parturition; vasculature of the uterus; uterine vascular adaptations to pregnancy; and maternal-fetal relationships and placental exchange. The chapters on menstruation and fetal circulation have been largely expanded. The last two thirds of the chapter on the physiologic basis of uterine-muscle therapy has been deleted as not being germane to the fundamental aspects of uterine physiology. The bibliography has been greatly expanded to more than twice the size of the first edition, now numbering nearly twenty-five hundred references. The illustrations are new except for a few of unusual merit. There are comprehensive indexes of authors and subjects. The type and printing are excellent. The use of a very heavy filled paper is not justified by the few plates needing that type of paper. Such plates could have been tipped into the text in their proper places as the color plate was. The total number of figures is only sixty-seven, including diagrams, charts and tracings. Books made of such paper, if wet, become as stiff as concrete. The work is recommended for all medical libraries and to physiologists, gynecologists and obstetricians.

Digitalis and Other Cardiotonic Drugs. By Eli R. Movitt, M.D., chief of medicine, Veterans Administration Hospital, Oakland, California. Second edition. 8°, cloth, 245 pp., with 20 illustrations. New York: Oxford University Press, 1949. $5.75. (*Oxford Medical Publications.*)

This second edition of a special monograph, first published in 1946, has been revised and brought up to date, incorporating the knowledge gained during the past three years. New material, especially on the clinical application of the cardiac glycosides, has been included. Lists of selected references are appended to the various chapters, and there are indexes of subjects and authors. The publishing is excellent.

La tubercolosi, problema medico-sociale. By Prof. Giovanni L'Eltore. 8°, paper, 187 pp., with 45 illustrations. Roma: Istituto di Medicina Sociale, 1949. (Collana di Studi Sui Problemi Medico-Sociali.)

This monograph discusses the social aspects of tuberculosis. It provides a great many tables and graphs on morbidity and mortality throughout the world and especially in Europe. Likewise it discusses, in full, incidence in relation to the other respiratory diseases, and to total morbidity, and antituberculosis measures, including sanatorium and postsanatorium care. This valuable work should be in all collections on the subject.

Health Instruction Yearbook 1949. Compiled by Oliver E. Byrd, Ed.D., M.D., professor of health education and director, Department of Hygiene, School of Education, Stanford University. With a foreword by Charles E. Smith, M.D., president, California State Board of Public Health, and dean, School of Public Health, University of California. 8°, cloth, 276 pp. Stanford, California: Stanford University Press, 1949. $3.50.

This annual issue of a well known yearbook contains 255 abstracts of selected articles covering the whole field of public health. Appended to the text is a bibliography citing the sources from which these articles were selected. There are author and subject indexes, and the book is well published. It is recommended for all collections on the subject and should prove invaluable to all workers in the field of public health.

The Eye and Its Diseases: By 92 international authorities. Edited by Conrad Berens, M.D., of the Ophthalmological Foundation, Inc., president, Snyder Ophthalmic Foundation, and president, American Academy of Ophthalmology and Otolaryngology. Second edition. 4°, cloth, 1092 pp., with 436 illustrations. Philadelphia: W. B. Saunders Company, 1949. $16.00.

This second edition of a book first published in 1936 has been thoroughly revised and the subject brought up to date. Chapters on illumination, physiologic chemistry and gonioscopy have been added. All sections have been revised. Ninety-two recognized authorities in their particular fields have written special chapters. There is a comprehensive index, and the volume is well published in every way. It is recommended for all medical libraries and to all persons interested in the subject.

Audiology: *The science of hearing. A developing professional specialty.* By Norton Canfield, M.D., associate professor of otolaryngology, Yale University School of Medicine. With a foreword by Gunnar Holmgren, professor of otolaryngology, Stockholm, Sweden. 8°, cloth, 45 pp. Springfield, Illinois: Charles C Thomas, 1949. $1.75.

This small monograph on a comparatively new subject comprises No. 51 of the *American Lecture Series*. The material covers the whole field of the subject in brief chapters. A rather extensive bibliography is appended to the text. The lack of an index is noted, but otherwise the monograph is well published.

Vitaminology: *The chemistry and function of the vitamins.* By Walter H. Eddy, Ph.D., scientific director, American Chlorophyll, Incorporated. 8°, cloth, 365 pp., with 60 tables. Baltimore: Williams and Wilkins Company, 1949. $6.00.

This new work on the vitamins by a recognized authority should prove valuable as a reference volume on the subject. It covers the whole field, devoting separate chapters to all the known vitamins. Numerous selective bibliographies are interpolated throughout the text. There is a good index, and the volume is well published and recommended for all medical libraries and to all persons interested in the subject.

X-Ray Treatment: *Its origin, birth and early history.* By Emil H. Grubbé, M.D. 8°, cloth, 153 pp., with 51 illustrations. St. Paul: The Bruce Publishing Company, 1949. $3.00.

This small volume on the history of x-ray treatment is especially devoted to the author's experience. The text is concluded with a list of physicians who pioneered in the field and became martyrs to the burns suffered during their work. In this connection it should be noted that Dr. Francis H. Williams, of Boston, was a pioneer in the field of roentgenology and wrote the first large treatise on the subject, in 1901. Previously Dr. Williams had collaborated with William Rollins on the physics of the x-ray. Dr. Williams, who was a graduate of the Massachusetts Institute of Technology, had the foresight to protect himself from the injurious effects of the rays and therefore did not become a martyr. This small book is well printed, but the lack of an index detracts greatly from its value.

Bring Out Your Dead: *The great plague of yellow fever in Philadelphia in 1793.* By J. H. Powell. 8°, cloth, 304 pp. Philadelphia: University of Pennsylvania Press, 1949. $3.75.

This semipopular story of a tragic event in America has been well written, and the material well organized. There is a good index, and the volume is well published and should be in all medical-history collections.

Selected Papers of Haven Emerson, A.M., M.D., D.S.M. Published on the occasion of his seventy-fifth birthday, October 19, 1949. 8°, cloth, 507 pp. Battle Creek, Michigan: W. K. Kellogg Foundation, 1949. $3.00.

Haven Emerson is well recognized as an outstanding authority in the field of public health. This volume brings together in one place thirty-five of his important papers, constituting a memorial of his seventy-fifth birthday. The first in the collection reprints his first paper on studies of the capsule of the kidney, presented in 1903 before the Association of American Physicians. Appended to the text is his personal bibliography of 159 titles and a list of the health or hospital surveys made by Dr. Emerson between 1920 and 1937. The volume is well published in every way and is notable for its use of a large, easily read type. The book is recommended to all medical libraries and to all persons interested in public health.

NOTICES

ANNOUNCEMENT

Dr. Benjamin C. Riggs announces the opening of offices at 330 Dartmouth Street, Boston, and 62 Main Street, Concord, for the practice of neuropsychiatry.

NEW ENGLAND SOCIETY OF ANESTHESIOLOGISTS

A regular meeting of the New England Society of Anesthesiologists will be held in the amphitheater of Building A, Boston University School of Medicine, 80 East Concord Street, Boston, on Tuesday, May 9, at 7:30 p.m. The scientific meeting will consist of a panel discussion of cases presented by the Anesthesia Study Committee. Dr. Meyer Saklad will be chairman.

The final meeting of the year of the New England Society of Anesthesiologists will be held in the Amphitheater of Building A, Boston University School of Medicine, 80 East Concord Street, Boston, on Tuesday, June 13, at 7:30 p.m.

The business meeting will be devoted to the annual meeting and election of officers.

The scientific meeting will consist of a symposium on anesthesia for ocular surgery, led by the following: Edwin B. Dunphy, M.D., Henry Willard Williams Professor of Ophthalmology, Harvard Medical School, and chief, Ophthalmological Service, Massachusetts Eye and Ear Infirmary; Paul A. Chandler, M.D., associate clinical professor in ophthalmology, Harvard Medical School, and senior surgeon, Massachusetts Eye and Ear Infirmary; and John H. Tucci, M.D., instructor in anesthesia, Harvard Medical School, and anesthetist, Massachusetts Eye and Ear Infirmary.

NEW ENGLAND STATES CHAPTER OF AMERICAN COLLEGE OF CHEST PHYSICIANS

A meeting of the New England States Chapter of the American College of Chest Physicians will be held in Harris Hall, Deaconess Hospital, Boston, on Wednesday, May 10, at 4 p.m. Drs. Richard H. Overholt and Leo Kenney will speak on the subject "Costoversion One-Stage Thoracoplasty."

A business meeting will be held at 5:30 p.m.

MASSACHUSETTS ACADEMY OF GENERAL PRACTICE

The spring meeting of the Massachusetts Academy of General Practice will be held in Boston on Wednesday, May 10, with headquarters at the Hotel Puritan, 360 Commonwealth Avenue, Boston.

PROGRAM

9:30 a.m. Business Meeting. Joseph H. Pratt Diagnostic Clinic.
10:30 a.m. to 1:00 p.m. Medical and Surgical Clinics under the direction of Dr. Samuel H. Proger. Joseph H. Pratt Diagnostic Clinic.
1:00 p.m. Registration. Hotel Puritan.
1:45 p.m. Luncheon. Hotel Puritan.
3:00 p.m. Anxiety and Symptom Formation. Dr. John M. Murray. Hotel Puritan.
4:00 p.m. Acute Abdomen, Crucial Signs and Symptoms. Dr. Leland S. McKittrick. Hotel Puritan.
5:00 p.m. Clinical Auscultation of the Heart. Dr. Samuel A. Levine. Hotel Puritan.
7:00 p.m. Dinner (guest speaker, Mr. Rudolph Elie, of the *Boston Herald*). Hotel Puritan.

BOSTON CITY HOSPITAL HOUSE OFFICERS' ASSOCIATION

A meeting of the Boston City Hospital House Officers' Association will be held in the Dowling Amphitheater of the Hospital on Thursday, May 18, at 7 p.m. Dr. George Crile, Jr., surgeon-in-chief of the Cleveland Clinic, will speak on the subject "The Present Status of the Treatment of Thyroid Diseases."

All interested members of the medical profession are invited to attend the lecture.

MASSACHUSETTS ASSOCIATION OF MEDICAL TECHNOLOGISTS

The annual meeting of the Massachusetts Association of Medical Technologists, sponsored by the Greater Boston Medical Technologists' Association, will be held at the Hotel Kenmore, Boston, on May 6 and 7.

NEW ENGLAND PEDIATRIC SOCIETY

A meeting of the New England Pediatric Society will be held in Hartford, Connecticut, on Wednesday, May 24. The afternoon clinical session will be held in the amphitheater of the Hartford Hospital, from 2:00 to 4.15 p.m. The evening speaker will be Dr. Joseph S. Collings, of Australia, who will discuss the subject "Changes in Patterns in Medical Care."

AMERICAN MEDICAL ASSOCIATION GOLF TOURNAMENT

The thirty-fourth golf tournament of the American Medical Association will be held on the two golf courses of the Olympic Golf Club, San Francisco, on Monday, June 26, the opening day of the annual session.

Fellows planning to participate should send, as soon as possible, their names, handicaps and sections in medicine in which they will register to Secretary William J. Burns, 2020 Olds Tower, Lansing 8, Michigan. The starting committee will help players from the different states to arrange games with opponents of similar handicaps, age and specialty.

"THIS IS YOUR DOCTOR"

On April 9 Station WLAW, Lawrence, Massachusetts, inaugurated thirteen consecutive weekly fifteen-minute programs from 3:15 to 3:30 p.m., entitled "This is Your Doctor."

These transcribed programs, sponsored by the Essex North District Medical Society in conjunction with the Greater Lawrence Retail Druggists' Association, dramatize the highlights in the lives of distinguished American physicians, emphasizing the importance and necessity of maintaining the traditional way of practicing unhampered American medicine.

RADIO BROADCASTS ON SOCIALIZED MEDICINE

Mr. Robert U. Johnson, executive secretary of the Independent Voters' Committee, will speak over station WBRK from 10:00 to 10:15 p.m., on Tuesday and Thursday evenings, for an indefinite period. Mr. Johnson is a vigorous opponent of the Welfare State and all that it implies. He is definitely interested in the fight against socialized medicine and has the backing of the Committee on Public Relations of Berkshire District Medical Society. Members are urged to listen to these broadcasts and to inform their friends and patients of them.

AMERICAN LARYNGOLOGICAL ASSOCIATION

The seventy-first annual meeting of the American Laryngological Association will be held at the Mark Hopkins Hotel, San Francisco, California, on May 23 and 24.

Members of the medical profession are cordially invited to attend.

KAPPA DELTA AWARD FOR RESEARCH IN ORTHOPEDIC SURGERY

A prize of $1000, donated by the Kappa Delta Sorority, is awarded annually by the American Academy of Orthopaedic Surgeons for the best research done in orthopedic surgery or related scientific field by a person in the United States. The second award, for the year 1950, will be presented at the eighteenth annual convention of the Academy in Chicago on January 31, 1951. Researchers interested in competing for this prize may obtain further information from Dr. R. Beverly Raney, Duke Hospital, Durham, North Carolina, chairman of the Award Committee for 1950.

SOCIETY MEETINGS AND CONFERENCES

October 3–May 19. Massachusetts Department of Mental Health. Postgraduate Seminar in Neurology and Psychiatry. Page 286, issue of August 18.

January 9–December 1. Laboratory Courses at Communicable Disease Center, Atlanta, Georgia. Page 950, issue of December 8.

May 6. Suffolk District Medical Society. Page 600, issue of April 13.

May 6 and 7. Massachusetts Association of Medical Technologists. Page 735.

May 8. New England Cardiovascular Society. Annual Meeting.

May 8. Henry Jackson Lecture. Page 688, issue of April 27.

May 9. New England Society of Anesthesiologists. Page 735.

May 10. New England States Chapter of American College of Chest Physicians. Page 735.

May 10. Massachusetts Academy of General Practice. Page 735.

May 10. Massachusetts Society of Examining Physicians. Page 528, issue of March 30.

May 10. Massachusetts Chapter of American Academy of General Practice. Page 528, issue of March 30.

May 10. Suffolk District Medical Society. Page 600, issue of April 13.

May 11. The Problems of Infertility. Dr. David Weintraub. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

May 12 and 13. Meetings at Texas Medical Center. Page 600, issue of April 13.

May 14–19. International and Fourth American Congress of Obstetrics and Gynecology. Page 160, issue of January 26.

May 16. The Springfield Hospital. Page 640, issue of April 20.

May 16–18. Massachusetts Medical Society. Annual Meeting. Hotel Statler, Boston.

May 17. Luncheon Meeting. Section on Diseases of the Eye, Ear Nose and Throat, Massachusetts Medical Society. Hotel Statler, Boston, 12:00 m.

May 18. The Springfield Hospital. Page 640, issue of April 20.

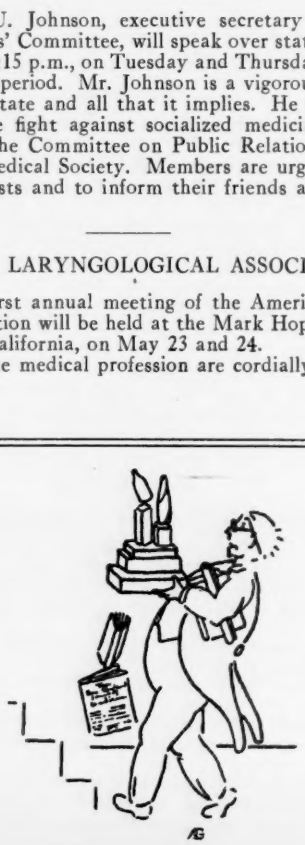
May 18. Boston City Hospital House Officers' Association. Page 735.

May 20. The Springfield Hospital. Page 640, issue of April 20.

May 22 and 23. National Research Council Committee on Human Reproduction. Page 488, issue of March 23.

May 23 and 24. American Laryngological Association. Notice above.

(Notices concluded on page xv)



When ordering needles cisternal,

Books, sponges or doses diurnal,

Dr. Wise always mentions

(Or has those intentions)

***The ads that he reads in the* Journal.**

11

NOTICES (*Concluded from page 736*)

MAY 24. New England Pediatric Society. Page 736.

MAY 24–26. American Association of Genito-Urinary Surgeons. Page 234, issue of February 9.

JUNE 12–14. American Neurological Association. Page 600, issue of April 13.

JUNE 13. New England Society of Anesthesiologists. Page 735.

JUNE 22–25. American College of Chest Physicians. Page 270, issue of February 16.

JUNE 24 AND 25. American Society for the Study of Sterility. Page 38, issue of January 5.

JUNE 24 AND 25. American Academy of Dental Medicine. Page 640, issue of April 20.

JUNE 26–30. American Physicians Art Association. Page 428, issue of March 16.

JUNE 26–30. American Medical Association. Annual Session. San Francisco, California.

JULY 17–22. International Congress for Scientific Research. Page xvii, issue of September 1.

AUGUST 21–26. International Society of Hematology. Page 888, issue of December 1.

AUGUST 28–SEPTEMBER 1. American Congress of Physical Medicine. Page 428, issue of March 16.

AUGUST 31–SEPTEMBER 7. World Federation for Mental Health. Page 306, issue of February 23.

SEPTEMBER 3–9. International Heart Congress. Page 160, issue of January 26.

SEPTEMBER 27–29. Mississippi Valley Medical Society. Page 991, issue of December 15.

OCTOBER 12–14. American Association of Blood Banks. Page 428, issue of March 16.

NOVEMBER 1. New England Obstetrical and Gynecological Society. Hotel Somerset, Boston.

JANUARY 31. American Academy of Orthopaedic Surgeons (see Kappa Delta Award for Research in Orthopedic Surgery). Page 736.

DISTRICT MEDICAL SOCIETY

SUFFOLK

MAY 6. Spring Dinner.

MAY 10. Annual Meeting.

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, MAY 11

FRIDAY, MAY 12

*9:00 a.m.–12:00 m. Combined Medical and Surgical Staff Rounds. Peter Bent Brigham Hospital.

12:00 m.–1:00 p.m. Clinicopathological Conference (Boston Floating Hospital). Joseph H. Pratt Diagnostic Hospital.

*1:30 p.m. Tumor Clinic. Out-Patient Department, Mount Auburn Hospital, Cambridge.

MONDAY, MAY 15

*11:30 a.m.–12:15 p.m. Chest X-Ray Conference. South End Health Unit, 57 East Concord Street, Boston. Dr. Cleaveland Floyd in charge.

*12:15–1:15 p.m. Clinicopathological Conference. Main Amphitheater, Peter Bent Brigham Hospital.

TUESDAY, MAY 16

*12:15–1:15 p.m. Clinicoroentgenological Conference. Peter Bent Brigham Hospital.

*1:30–2:30 p.m. Pediatric Rounds. Burnham Memorial Hospital for Children, Massachusetts General Hospital.

WEDNESDAY, MAY 17

*11:00 a.m.–12:00 m. Medical Rounds. Children's Medical Center.

*12:00 m. X-Ray Conference. Margaret Jewett Hall, Mount Auburn Hospital, Cambridge.

*12:00 m.–1:00 p.m. Clinicopathological Conference (Children's Medical Center). Amphitheater, Peter Bent Brigham Hospital.

*2:00–3:00 p.m. Combined Clinic of all Medical Center Services. Amphitheater, Children's Hospital.

*Open to the medical profession.

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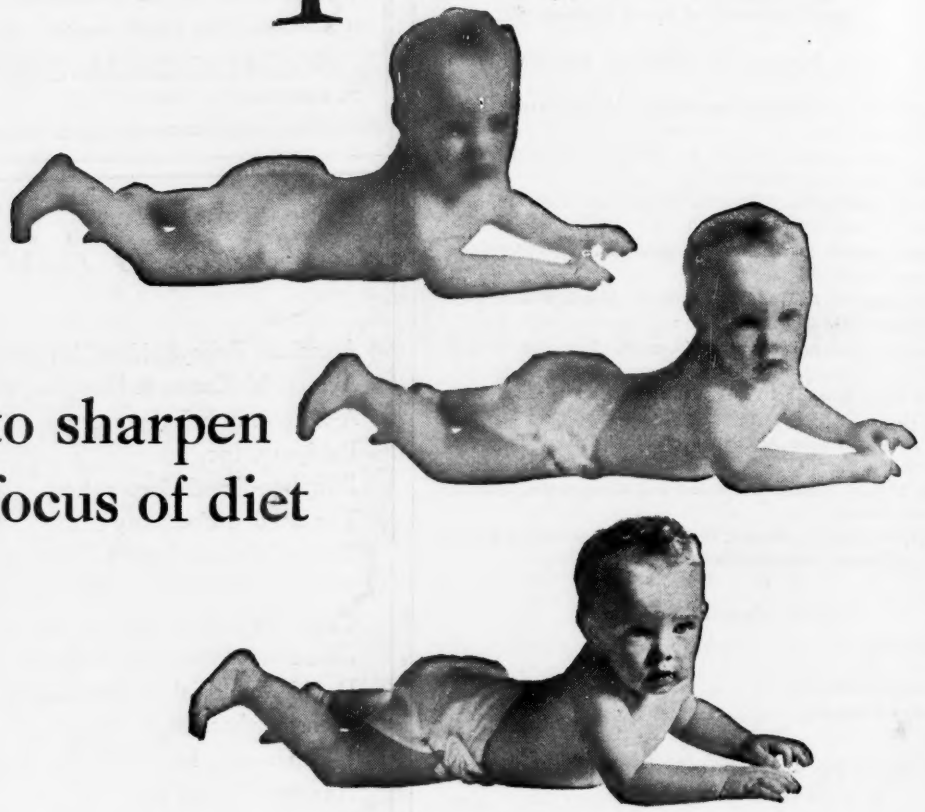
Flexible Formula



...to sharpen the focus of diet

WHEN an infant's diet is not formulated to his exact needs, it is like a picture out of focus. For an individualist, the basic formula must be *flexible* to meet the changing needs of the moment—to bring the diet "into focus."

Dextri-Maltose* has been preferred by two generations of physicians because of its exceptional flexibility in formulas using whole or evaporated milk. Quantities of this carbohydrate can be varied at will with the varying caloric requirements of the individual infant; and Dextri-Maltose is available in *five* forms to meet certain clinical conditions without disturbing the feeding routine.

Not too sweet, readily soluble and easy to use, Dextri-Maltose is highly digestible and slowly absorbed. No other carbohydrate for infant feeding enjoys so authoritative a background of clinical experience.

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DEXTRI-MALTOSE

DEXTRI-MALTOSE NO. 1—with 2% sodium chloride • DEXTRI-MALTOSE NO. 2—Plain • DEXTRI-MALTOSE NO. 3—with 3% Potassium Bicarbonate • DEXTRI-MALTOSE WITH YEAST EXTRACT AND IRON • PECTIN-AGAR IN DEXTRI-MALTOSE.

Descriptive literature on request